SYSTEM:OS - DIALOG OneSearch
File 155:MEDLINE(R) 1966-2002/Mar W5
File 5:Biosis Previews(R) 1969-2002/Mar W5 (c) 2002 BIOSIS

Set	Items	Description
S1	276504	CHANNEL?
S2	5137	CONOTOXIN
S3	4321	S1 AND S2
S4	37107	OMEGA
S5	3930	S4 AND S3
S6	91208	EPILEPSY
S7	29	S5 AND S6
S8	19	RD (unique items)
S9	478825	PAIN OR INFLAMMATION
S10	72	S9 AND S5
S11	56	RD (unique items)
S12	287570	HYPOXIA OR ANOXIA OR ISCHEMIA
S13	113	S12 AND S5
S14	72	RD (unique items)
S15	1	J410 OR J411 OR J413 OR J414
S16	0	J(W)(410 OR 411 OR 413 OR 414)
S17	37	ARENATUS
S18	0	S2 AND S17
S19	1	AURISIACUS
S20	109	BULLATUS
S21	0	S2 AND S20
S22	2	CARACTERISTICUS
S23	1225	CATUS
S24	0	S2 AND S23
S25	2	CIRCUMCISUS
S26	87	CONSORS
S27	9	S2 AND S26
S28	6	RD (unique items)
S29	873	DALLI OR DISTANS
S30	0	S2 AND S29
S31	22	ERMINEUS
S32	7	S2 AND S31
S33	6	RD (unique items)
S34	273	GEOGRAPHUS
S35	156	S2 AND S34
S36	80	S4 AND S35
S37	58	RD (unique items)
S38	1	LATERCULATUS
S39	204	LEOPARDUS OR LYNCEUS
S40	0	S2 AND S39
S41	320	MAGUS
S42	207	S2 AND S41
S43	183	S4 AND S42
S44	172	RD (unique items)
S45	3852	MILES
S46	0	S2 AND S45
S47	2644	MONACHUS OR OBSCURUS
S48	2	S2 AND S47
S49	834	PULICARIUS OR PURPURASCENS
S50	27	S2 AND S49
S51	17	RD (unique items)
S52	872	RADIATUS
S53	4	S2 AND S52
S54	8629	RATTUS
S55	0	S2 AND S54
S56	21	STERCUSMUSCARUM
S57	0	S2 AND S56
S58	2213	STRIATUS
S59	19	S2 AND S58
S60	13	RD (unique items)
S61	64	STRIOLATUS
S62	0	S2 AND S61
S63	5346	TEXTILE
S64	22	S2 AND S63
S65	16	RD (unique items)
S66	2046	TULIPA OR VIOLA
S67	8	S2 AND S66
S68	5	RD (unique items)
S69	32	PULICARIUS
S70	0	S2 AND S69

8/6/1 (Item 1 from file: 155) 11553531 21297885 PMID: 11403944
Levetiracetam inhibits the high-voltage-activated Ca(2+) current in pyramidal neurones of rat hippocampal slices. Jun 22 2001

8/6/2 (Item 2 from file: 155) 10967854 20565846 PMID: 11113304
Seizures and neurodegeneration induced by 4-aminopyridine in rat hippocampus in vivo: role of glutamate- and GABA-mediated neurotransmission and of ion channels . 2000

8/6/3 (Item 3 from file: 155) 10874656 20496841 PMID: 11040345
Opposite effects of T- and L-type Ca(2+) channels blockers in generalized absence epilepsy . Oct 20 2000

8/6/4 (Item 4 from file: 155) 10822081 20503403 PMID: 11051119
Modulation of calcium channels by group I and group II metabotropic glutamate receptors in dentate gyrus neurons from patients with temporal lobe epilepsy . Oct 2000

8/6/5 (Item 5 from file: 155) 10592778 20193910 PMID: 10727713
Inhibition of voltage-gated calcium channels by fluoxetine in rat hippocampal pyramidal cells. Apr 3 2000

8/6/6 (Item 6 from file: 155) 10469388 20083611 PMID: 10617321
Valproic acid intensifies epileptiform activity in the hippocampal pyramidal neurons. Dec 30 1999

8/6/7 (Item 7 from file: 155) 10323264 98432498 PMID: 9761331
Voltage-dependent Ca2+ curr

8/6/8 (Item 8 from file: 155) 10310485 98370968 PMID: 9705268
Altered expression and assembly of N-type calcium channel alpha1B and beta subunits in epileptic lethargic (lh/lh) mouse. Aug 21 1998

8/6/9 (Item 9 from file: 155) 09974736 98429303 PMID: 9758336
Differential expression and association of calcium channel subunits in development and disease. Aug 1998

8/6/10 (Item 10 from file: 155) 09464157 97238297 PMID: 9084617
Properties of voltage-activated Ca2+ currents in acutely isolated human hippocampal granule cells. Mar 1997

8/6/11 (Item 11 from file: 155) 09226126 97005922 PMID: 8853221
Behavioural and anticonvulsant effects of Ca2+ channel toxins in DBA/2 mice. Jul 1996

8/6/12 (Item 12 from file: 155) 09193979 96388222 PMID: 8795623
Enhanced fast synaptic transmission and a delayed depolarization induced by transient potassium current blockade in rat hippocampal slice as studied by optical recording. Sep 15 1996

8/6/13 (Item 13 from file: 155) 09037494 96427796 PMID: 8831112
Lamotrigine inhibits Ca2+ currents in cortical neurons: functional implications. Jun 20 1996

8/6/14 (Item 14 from file: 155) 08858277 94306189 PMID: 8032931
The voltage-sensitive Ca2+ channel (VSCC) antagonists omega -Aga-IVA and omega -CTX-MVIIC inhibit spontaneous epileptiform discharges in the rat cortical wedge. Apr 18 1994

8/6/15 (Item 15 from file: 155) 07074921 93286688 PMID: 8389832
Calcium currents in acutely isolated human neocortical neurons. May 1993

8/6/16 (Item 1 from file: 5) 13159829 BIOSIS NO.: 200100366978
Pharmacological discrimination between effects of carbamazepine on hippocampal basal, Ca2+ - and K+-evoked serotonin release. 2001

8/6/17 (Item 2 from file: 5) 12881264 BIOSIS NO.: 200100088413
Mechanisms of nicotine-induced (3H)norepinephrine release in human cerebral cortex slices. 2000

8/6/18 (Item 3 from file: 5) 10470115 BIOSIS NO.: 199699091260
Cholinergic-dependent plateau potential in hippocampal CA1 pyramidal neurons. 1996

8/6/19 (Item 4 from file: 5) 09583596 BIOSIS NO.: 199598038514
Voltage-sensitive calcium channel development in epileptic DBA/2J mice suggests altered presynaptic function. 1994

8/7/2 (Item 2 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10967854 20565846 PMID: 11113304
Seizures and neurodegeneration induced by 4-aminopyridine in rat hippocampus in vivo: role of glutamate- and GABA-mediated neurotransmission and of ion channels .
Pena F; Tapia R
Departamento de Neurociencias, Instituto de Fisiologia Celular,Universidad Nacional Autonoma de Mexico, AP 70-253, 04510, D.F., MexicoCity, Mexico.
Neuroscience (UNITED STATES) 2000, 101 (3) p547-61, ISSN 0306-4522 Journal Code: NZR Languages: ENGLISH
Document type: Journal Article Record type: Completed
Infusion of the K(+) channel blocker 4-aminopyridine in the hippocampus induces the release of glutamate, as well as seizures and neurodegeneration. Since an imbalance between excitation and inhibition, as well as alterations of ion channels , may be involved in these effects of 4-aminopyridine, we have studied whether they are modified by drugs that block glutamatergic transmission or ion channels , or drugs that potentiate GABA-mediated transmission. The drugs were administered to anesthetized rats subjected to intrahippocampal infusion of 4-aminopyridine through microdialysis probes, with simultaneous collection of dialysis perfusates and recording of the electroencephalogram, and subsequent histological analysis. Ionotropic glutamate receptor antagonists clearly diminished the intensity of seizures and prevented the neuronal damage, but did not alter substantially the enhancement of extracellular glutamate induced by 4-aminopyridine. None of the drugs facilitating GABA-mediated transmission, including uptake blockers, GABA-transaminase inhibitors and agonists of the A-type receptor, was able

to reduce the glutamate release, seizures or neuronal damage produced by 4-aminopyridine. In contrast, nipecotate, which notably increased extracellular levels of the amino acid, potentiated the intensity of seizures and the neurodegeneration. GABA(A) receptor antagonists partially reduced the extracellular accumulation of glutamate induced by 4-aminopyridine, but did not exert any protective action. Tetrodotoxin largely prevented the increase of extracellular glutamate, the electroencephalographic epileptic discharges and the neuronal death in the CA1 and CA3 hippocampal regions. Valproate and carbamazepine, also Na(+) channel blockers that possess general anticonvulsant action, failed to modify the three effects of 4-aminopyridine studied. The N-type Ca(2+) channel blocker omega - conotoxin , the K(+) channel opener diazoxide, and the non-specific ion channel blocker riluzole diminished the enhancement of extracellular glutamate and slightly protected against the neurodegeneration. However, the two former compounds did not antagonize the 4-aminopyridine-induced epileptiform discharges, and riluzole instead markedly increased the intensity and duration of the disharges. Moreover, at the highest dose tested (8mg/kg, i.p.), riluzole caused a 75% mortality of the rats.We conclude that 4-aminopyridine stimulates the release of glutamate from nerve endings and that the resultant augmented extracellular glutamate is directly related to the neurodegeneration and is involved in the generation of epileptiform discharges through the concomitant overactivation of glutamate receptors. Under these conditions, a facilitated GABA-mediated transmission may paradoxically boost neuronal hyperexcitation. Riluzole, a drug used to treat amyotrophic lateral sclerosis, seems to be toxic when combined with neuronal hyperexcitation. Record Date Created: 20010112

8/7/3 (Item 3 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10874656 20496841 PMID: 11040345
Opposite effects of T- and L-type Ca(2+) channels blockers in generalized absence epilepsy .
van Luijtelaar G; Wiadema D; Elants C; Scheenen W
NICI, Department of Comparative and Physiological Psychology, Psychology Laboratory, Nijmegen University, PO Box 9104, 6500 HE, Nijmegen, Netherlands. luitelaar@nici.kun.nl
European journal of pharmacology (NETHERLANDS) Oct 20 2000, 406 (3) p381-9, ISSN 0014-2999 Journal Code: EN6 Languages: ENGLISH Document type: Journal Article Record type: Completed
The role of the T-type Ca(2+) channel blocker, ethosuximide, the L-type Ca(2+) channel blocker, nimodipine and L-type Ca(2+) channel opener, BAY K8644 (1,4 Dihydro-2, 6-dimethyl-5-nitro-4-[trifluoromethyl)-phenyl]-3- pyridine carboxylic acid methyl ester), was investigated on spike-wave discharges in WAG/Rij rats. This strain is considered as a genetic model for generalized absence epilepsy . A dose-dependent decrease in the number of spike-wave discharges was found after i.c.v. ethosuximide, an increase after i.p. nimodipine and a decrease after i.c.v. BAY K8644. BAY K8644 was also able to antagonise the effects of nimodipine. Preliminary data were obtained with two conotoxins, MVIIC and GVIA, which block P/Q-type and N-type Ca(2+) channels , respectively. Only after i.c.v. administration of omega - conotoxin GVIA were the number and duration of spike-wave discharges reduced, but animals showed knock-out lying. The latter suggests behavioural or toxic effects and that the decrease in spike-wave activity cannot unequivocally be attributed to blockade of N-type Ca(2+) channels .It can be concluded that T- and L-type Ca(2+) channel blockers show opposite effects on spike-wave discharges. Furthermore, these effects are difficult to explain in terms of a model for spindle burst activity in thalamic relay cells proposed by McCormick and Bal [Sleep and arousal: thalamocortical mechanisms. Record Date Created: 20001211

8/7/4 (Item 4 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10822081 20503403 PMID: 11051119
Modulation of calcium channels by group I and group II metabotropic glutamate receptors in dentate gyrus neurons from patients with temporal lobe epilepsy .
Schumacher TB; Beck H; Steffens R; Blumcke I; Schramm J; Elger CE; Steinhauser C
Department of Neurosurgery, University of Bonn, Germany.
Epilepsia (UNITED STATES) Oct 2000, 41 (10) p1249-58, ISSN 0013-9580 Journal Code: EIX Languages: ENGLISH Document type: Journal Article Record type: Completed
PURPOSE: Metabotropic glutamate receptors (mGluRs) might be promising new drug targets for the treatment of epilepsy because the expression of certain mGluRs is regulated in epilepsy and because activation of mGluRs results in distinctive anti- and proconvulsant effects. Therefore, we examined how mGluR activation modulates high-voltage-activated (HVA) Ca2+ channels . METHODS: Whole-cell patch-clamp recordings were obtained from granule cells and interneuron-like cells acutely isolated from the dentate gyrus of patients with pharmacoresistent temporal lobe epilepsy . RESULTS: Agonists selective for either group I or group II mGluRs rapidly and reversibly reduced HVA currents in most dentate gyrus cells. These modulatory effects were inhibited by the respective group I and group II mGluR antagonists. The specific Ca2+ channel antagonists nifedipine and omega - conotoxin GVIA potently occluded the effects of group I and II mGluR agonists, respectively, indicating that group I mGluRs acted on L-type channels and group II mGluRs affected N-type channels . About two thirds of the responsive neurons were sensitive either to group I or group II mGluRs, whereas a minority of cells showed effects to agonists of both groups, indicating a variable mGluR expression pattern. CONCLUSIONS: Group I and group II mGluRs are expressed in human dentate gyrus neurons and modulate L- and N-type HVA channels , respectively. The data shed light on the possible cellular sequelae of the mGluR1 upregulation observed in human epileptic dentate gyrus as well as on possible mGluR-mediated anticonvulsant mechanisms. Record Date Created: 20001026

8/7/7 (Item 7 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10323264 98432498 PMID: 9761331
Voltage-dependent Ca2+ currents in epilepsy .
Beck H; Steffens R; Elger CE; Heinemann U
Department of Experimental Epileptology, University of Bonn Medical Center, Germany. heinz@mailer.meb.uni-bonn.de
Epilepsy research (NETHERLANDS) Sep 1998, 32 (1-2) p321-32, ISSN 0920-1211 Journal Code: EMA Languages: ENGLISH Document type: Journal Article Record type: Completed
Voltage-dependent Ca2+ channels (VCCs) represent one of the main routes of Ca2+ entry into neuronal cells. Changes in intracellular Ca2+ dynamics and homeostasis can cause long-lasting cellular changes via activation of different Ca2+ dependent signalling pathways. We have investigated the properties of VCCs in human hippocampal dentate granule cells (DGCs) using the whole-cell configuration of the patch-clamp method. Classical high-threshold Ca2+ currents were composed mainly of omega - CgTx-sensitive N-type and nifedipine-sensitive L-type currents that were present in similar proportions. In addition, a Ca2+ current component that was sensitive to low concentrations of Ni2+, but not to nifedipine or omega - conotoxin GVIA (omega - CgTx GVIA) was present. This latter component showed a half-maximal inactivation at more hyperpolarized potentials than high-threshold currents and a more rapid time-dependent inactivation. This current was termed T-type Ca2+ current. Current components with similar pharmacological and kinetic characteristics could be elicited in acutely isolated control rat DGCs. The current density of high threshold and T-type Ca2+ components was significantly larger in human DGCs and in the kainate model compared to DGCs isolated from adult control rats. These differences in current density were not accompanied by parallel differences in the voltage-dependence of VCCs. Taken together, these data suggest that an up-regulation of Ca2+ current density may occur in hippocampal epileptogenesis without consistent changes in Ca2+ current properties. Record Date Created: 19981229

8/7/9 (Item 9 from file: 155) DIALOG(R)File 155:MEDLINE(R)
09974736 98429303 PMID: 9758336
Differential expression and association of calcium channel subunits in development and disease.
McEnery MW; Vance CL; Begg CM; Lee WL; Choi Y; Dubel SJ
Department of Physiology and Biophysics, Case Western Reserve University, School of Medicine, Cleveland, Ohio 44106-4970, USA.
Journal of bioenergetics and biomembranes (UNITED STATES) Aug 1998, 30 (4) p409-18, ISSN 0145-479X Journal Code: HIO Languages: ENGLISH Document type: Journal Article; Review; Review, Tutorial Record type: Completed
Voltage-gated calcium channels (VDCC) are essential to neuronal maturation and differentiation. It is believed that important signaling information is encoded by VDCC-mediated calcium influx that has both spatial and temporal components. VDCC are multimeric complexes comprised of a pore-forming alpha1 subunit and auxiliary beta and alpha2/delta subunits. Changes in the fractional contribution of distinct calcium conductances to the total calcium current have been noted in developing and differentiating neurons. These changes are anticipated to reflect the differential expression and localization of the pore-forming alpha1 subunits. However, as in vitro studies have established that beta regulates the channel properties and targeting of alpha1, attention has been directed toward the developmental expression and assembly of beta isoforms. Recently, changes in the beta component of the omega - conotoxin GVIA (CTX)-sensitive N-type VDCC have indicated differential assembly of alpha1B with beta in postnatal rat brain. In addition, unique properties of beta4 have been noted with respect to its temporal pattern of expression and incorporation into N-type VDCC complexes. Therefore, the expression and assembly of specific alpha1/beta complexes may reflect an elaborate cellular strategy for regulating VDCC diversity. The importance of these developmental findings is bolstered by a recent study which identified mutations in the beta4 as the molecular defect in the mutant epileptic mouse (lethargic; lh/lh). As beta4 is normally expressed in both forebrain and cerebellum, one may consider the impact of the loss of beta4 upon VDCC assembly and activity. The importance of the beta1b and beta4 isoforms to calcium channel maturation and assembly is discussed. (83 Refs.) Record Date Created: 19990107

8/7/11 (Item 11 from file: 155) DIALOG(R)File 155:MEDLINE(R)
09226126 97005922 PMID: 8853221
Behavioural and anticonvulsant effects of Ca2+ channel toxins in DBA/2 mice.
Jackson HC; Scheideler MA
Health Care Discovery, Novo Nordisk A/S, Malov, Denmark.
Psychopharmacology (GERMANY) Jul 1996, 126 (1) p85-90, ISSN 0033-3158 Journal Code: QGI Languages: ENGLISH Document type: Journal Article Record type: Completed
This study investigated the behavioural and anticonvulsant effects of voltage-sensitive calcium channel blockers in DBA/2 mice. Omega - Conotoxin MVIIC (0.1, 0.3 micrograms ICV/mouse) and omega -agatoxin IVA (0.1, 0.3, 1 micrograms ICV), which act predominantly at P- and/or Q-type calcium channels , prevented clonic and tonic sound-induced seizures in this animal model of reflex epilepsy (ED50 values with 95% confidence limits for protection against clonic sound-induced seizures were 0.09 (0.04-0.36) micrograms ICV and 0.09 (0.05-0.15) micrograms ICV respectively and against tonic seizures 0.07 (0.03-0.16) micrograms ICV and 0.08 (0.04-0.13) micrograms ICV, respectively). The N-type calcium channel antagonists omega - conotoxin GVIA and omega - conotoxin MVIIA were also tested in this model. Omega - Conotoxin GVIA was anticonvulsant in DBA/2 mice, but only at high doses (3 micrograms ICV prevented tonic seizures in 60% of the animals; 10 micrograms ICV prevented clonic seizures in 60% and tonic seizures in 90% of the animals), whereas omega - conotoxin MVIIA did not inhibit sound-induced seizures in doses up to 10 micrograms ICV. Both omega - conotoxin GVIA and omega - conotoxin MVIIA induced an intense shaking

syndrome in doses as low as 0.1 microgram ICV, whereas omega - conotoxin MVIIC and omega -agatoxin IVA did not produce shaking at any of the doses examined. Finally, omega - conotoxin GI (0.01-1 microgram ICV) and alpha- conotoxin SI (0.3-30 micrograms ICV), which both act at acetylcholine nicotinic receptors, were not anticonvulsant and did not induce shaking in DBA/2 mice. These results confirm that blockers of N- and P-/Q-type calcium channels produce different behavioural responses in animals. The anticonvulsant effects of omega - conotoxin MVIIC and omega -agatoxin IVA in DBA/2 mice are consistent with reports that P- and/or Q-type calcium channel blockers inhibit the release of excitatory amino acids and are worthy of further exploration. Record Date Created: 19961223

8/7/14 (Item 14 from file: 155) DIALOG(R)File 155:MEDLINE(R)
08858277 94306189 PMID: 8032931
The voltage-sensitive Ca2+ channel (VSCC) antagonists omega -Aga-IVA and omega -CTX-MVIIC inhibit spontaneous epileptiform discharges in the rat cortical wedge.
Robichaud LJ; Wurster S; Boxer PA
Department of Neuroscience Pharmacology, Parke-Davis Pharmaceutical Research, Division of Warner-Lambert Co., Ann Arbor, MI 48105.
Brain research (NETHERLANDS) Apr 18 1994, 643 (1-2) p352-6, ISSN 0006-8993 Journal Code: B5L Languages: ENGLISH Document type: Journal Article Record type: Completed
The ability of VSCC antagonists to modulate excitatory amino acid (EAA) release was evaluated by measuring N-methyl-D-aspartate (NMDA) receptor-dependent spontaneous epileptiform discharges in rat cortical wedges. The N-type channel blocker omega -CTX-GVIA (300 nM) was ineffective. The P-type channel blocker omega -Aga-IVA at 300 nM reduced the frequency of discharges by 63%, while 300 nM omega -CTX-MVIIC reduced the frequency by 35%. These results coupled with the absence of NMDA antagonism by omega -Aga-IVA or omega -CTX-MVIIC in the cortical wedge suggest that the VSCCs blocked by these toxins are primarily responsible for mediating impulse dependent EAA release in the rat neocortex. Record Date Created: 19940812
Tags: Animal; Comparative Study; In Vitro; Male
Descriptors: Calcium Channel Blockers--pharmacology--PD; *Cerebral Cortex--physiology--PH; * Epilepsy ; *Peptides--pharmacology--PD; *Receptors, N-Methyl-D-Aspartate--physiology--PH; *Spider Venoms --pharmacology--PD; Cerebral Cortex--drug effects--DE; Cerebral Cortex --physiopathology--PP; Electrophysiology--methods--MT; Evoked Potentials --drug effects--DE; Evoked Potentials--physiology--PH; Rats; Rats, Wistar; Receptors, N-Methyl-D-Aspartate--antagonists and inhibitors--AI; Time Factors; omega -Agatoxin IVA CAS Registry No.: 0 (Calcium Channel Blockers); 0 (Peptides); 0 (Receptors, N-Methyl-D-Aspartate); 0 (Spider Venoms); 0 (omega-Agatoxin IVA); 147794-23-8 (omega-co

11/6/1 (Item 1 from file: 155) 12909597 21866200 PMID: 11877344
Algogen-specific pain processing in mouse spinal cord: differential involvement of voltage-dependent Ca(2+) channels in synaptic transmission. Mar 5 2002

11/6/2 (Item 2 from file: 155) 12730495 21628673 PMID: 11754873
Biochemical and pharmacological characterization of the venom of the black scorpion Heterometrus spinifer. Jan 1 2002

11/6/3 (Item 3 from file: 155) 11788623 21320133 PMID: 11426839
Antinociceptive action of amlodipine blocking N-type Ca2+ channels at the primary afferent neurons in mice. May 11 2001

11/6/4 (Item 4 from file: 155) 11749214 21428771 PMID: 11543953
Substance P and neurokinin A mediate sensory synaptic transmission in young rat dorsal horn neurons. Jul 1 2001

11/6/5 (Item 5 from file: 155) 11661155 21387571 PMID: 11496122
Differential nociceptive responses in mice lacking the alpha(1B) subunit of N-type Ca(2+) channels . Aug 8 2001

11/6/6 (Item 6 from file: 155) 11631830 21299808 PMID: 11406336
Role of calcium channels in the spinal transmission of nociceptive information from the mesentery. Jul 2001

11/6/7 (Item 7 from file: 155) 11599269 21380241 PMID: 11487594
Differential involvement of conotoxin -sensitive mechanisms in neurogenic vasodilatation responses: effects of age. Aug 2001

11/6/8 (Item 8 from file: 155) 11552881 21276249 PMID: 11382408
Distribution of various calcium channel alpha(1) subunits in murine DRG neurons and antinociceptive effect of omega - conotoxin SVIB in mice. Jun 8 2001

11/6/9 (Item 9 from file: 155) 11485543 21223391 PMID: 11323145
Effects of spinally delivered N- and P-type voltage-dependent calcium channel antagonists on dorsal horn neuronal responses in a rat model of neuropathy. May 2001

11/6/10 (Item 10 from file: 155) 11198570 21104047 PMID: 11160500
Axotomy- and autotomy-induced changes in Ca2+ and K+ channel currents of rat dorsal root ganglion neurons. Feb 2001

11/6/11 (Item 11 from file: 155) 10914159 20578057 PMID: 11133635
The combined effects of N-type calcium channel blockers and morphine on A delta versus C fiber mediated nociception. Jan 2001

11/6/12 (Item 12 from file: 155) 10874394 20516143 PMID: 11060815
An evaluation of intrathecal ziconotide for the treatment of chronic pain . Oct 2000

11/6/13 (Item 13 from file: 155) 10663729 20283152 PMID: 10822250
Structure-activity relationships of omega -conotoxins at N-type voltage-sensitive calcium channels . Mar-Apr 2000

11/6/14 (Item 14 from file: 155) 10580919 20176956 PMID: 10714496
Design and biological evaluation of non-peptide analogues of omega - conotoxin MVIIA. Feb 21 2000

11/6/15 (Item 15 from file: 155) 10579072 20258196 PMID: 10797861
Effects of intrathecal L- and N-type calcium channel blockers on the antinociception evoked by opioid agonists in the rat tail flick test. 1999

11/6/16 (Item 16 from file: 155) 10520046 20091027 PMID: 10623483
Benzylamine-related compounds stimulate rat vas deferens neurotransmission and potentiate memory in the mouse acting as potassium channel blockers. Feb 2000

11/6/17 (Item 17 from file: 155) 10485779 20106610 PMID: 10643816
Prosaptide D5 reverses hyperalgesia: inhibition of calcium channels through a pertussis toxin-sensitive G-protein mechanism in the rat. Jan 7 2000

11/6/18 (Item 18 from file: 155) 10344134 99423872 PMID: 10493735
delta opioid receptor modulation of several voltage-dependent Ca(2+) currents in rat sensory neurons. Oct 1 1999

11/6/19 (Item 19 from file: 155) 10331997 99158525 PMID: 10051216
Nerve injury increases an excitatory action of neuropeptide Y and Y2-agonists on dorsal root ganglion neurons. Mar 1999

11/6/20 (Item 20 from file: 155) 10322509 99042047 PMID: 9822729
Axotomy reduces the effect of analgesic opioids yet increases the effect of nociceptin on dorsal root ganglion neurons. Dec 1 1998

11/6/21 (Item 21 from file: 155) 10322497 99019747 PMID: 9801368
Depolarization stimulates initial calcitonin gene-related peptide expression by embryonic sensory neurons in vitro. Nov 15 1998

11/6/22 (Item 22 from file: 155) 10309071 98290368 PMID: 9628404
Spinal application of omega - conotoxin GVIA, an N-type calcium channel antagonist, attenuates enhancement of dorsal spinal neuronal responses caused by intra-articular injection of mustard oil in the rat. May 1998

11/6/23 (Item 23 from file: 155) 10308957 98288150 PMID: 9622667
Differential effects of intrathecally administered N- and P-type voltage-sensitive calcium channel blockers upon two models of experimental mononeuropathy in the rat. Jun 1 1998

11/6/24 (Item 24 from file: 155) 10295869 98081532 PMID: 9421179
Omega -agatoxin IVA, a P-type calcium channel antagonist, reduces nociceptive processing in spinal cord neurons with input from the inflamed but not from the normal knee joint--an electrophysiological study in the rat in vivo. Oct 1997

11/6/25 (Item 25 from file: 155) 10290745 97404278 PMID: 9262364
Differential effects of omega - conotoxin GVIA, nimodipine, calmidazolium and KN-62 injected intrathecally on the antinociception induced by beta-endorphin, morphine and [D-Ala2,N-MePhe4,Gly-ol5]-enkephali n administered intracerebroventricularly in the mouse. Aug 1997

11/6/26 (Item 26 from file: 155) 10215543 99306960 PMID: 10375670
Effects of adrenergic stimulus on the activities of Ca2+ and K+ channels of dorsal root ganglion neurons in a neuropathic pain model. Jun 19 1999

11/6/27 (Item 27 from file: 155) 10109296 98398342 PMID: 9729273
Effect of subcutaneous administration of calcium channel blockers on nerve injury-induced hyperalgesia. Aug 10 1998

11/6/28 (Item 28 from file: 155) 09983819 99006668 PMID: 9792182
Pharmacotherapeutic potential of omega - conotoxin MVIIA (SNX-111), an N-type neuronal calcium channel blocker found in the venom of Conus magus. Nov 1998

11/6/29 (Item 29 from file: 155) 09950770 99003408 PMID: 9786981
Differences in Ca2+ channels governing generation of miniature and evoked excitatory synaptic currents in spinal laminae I and II. Nov 1 1998

11/6/30 (Item 30 from file: 155) 09577589 97426533 PMID: 9278519
Serotonergic inhibition of the T-type and high voltage-activated Ca2+ currents in the primary sensory neurons of Xenopus larvae. Sep 15 1997

11/6/31 (Item 31 from file: 155) 09524291 97138888 PMID: 8985872
Effects of N- and L-type calcium channel antagonists on the responses of nociceptive spinal cord neurons to mechanical stimulation of the normal and the inflamed knee joint. Dec 1996

11/6/32 (Item 32 from file: 155) 09473922 98030309 PMID: 9365027
Effects of intrathecal injection of nimodipine, omega - conotoxin GVIA, calmidazolium, and KN-62 on the antinociception induced by cold water swimming stress in the mouse. Aug 29 1997

11/6/33 (Item 33 from file: 155) 09470579 97355282 PMID: 9211477
Blockade of calcium channels can prevent the onset of secondary hyperalgesia and allodynia induced by intradermal injection of capsaicin in rats. Jun 1997

11/6/34 (Item 34 from file: 155) 09462329 97213253 PMID: 9060018
Blockade of spinal N- and P-type, but not L-type, calcium channels inhibits the excitability of rat dorsal horn neurones produced by subcutaneous formalin inflammation . Jan 1997

11/6/35 (Item 35 from file: 155) 09222381 96416732 PMID: 8819527
Spinal morphine/clonidine antinociceptive synergism: involvement of G proteins and N-type voltage-dependent calcium channels . Sep 1996

11/6/36 (Item 36 from file: 155) 08991668 96264291 PMID: 8848159
Mechanism of prostaglandin E2-induced substance P release from cultured sensory neurons. Jan 1996

11/6/37 (Item 37 from file: 155) 08899552 95363679 PMID: 7636726
Synthetic omega -conopeptides applied to the site of nerve injury suppress neuropathic pains in rats. Aug 1995

11/6/38 (Item 38 from file: 155) 08891764 95114897 PMID: 7815344
Calcium modulation of morphine analgesia: role of calcium channels and intracellular pool calcium. Jan 1995

11/6/39 (Item 39 from file: 155) 08459804 95114899 PMID: 7815346
Modulation of cannabinoid-induced antinociception after intracerebroventricular versus intrathecal administration to mice: possible mechanisms for interaction with morphine. Jan 1995

11/6/40 (Item 40 from file: 155) 08181940 94285058 PMID: 8014856
Role of voltage-dependent calcium channel subtypes in experimental tactile allodynia. Jun 1994

11/6/41 (Item 41 from file: 155) 07841248 92183705 PMID: 1724655
Ruthenium red and capsaicin induce a neurogenic inflammatory response in the rabbit eye: effects of omega - conotoxin GVIA and tetrodotoxin. Dec 17 1991

11/6/42 (Item 42 from file: 155) 07704808 92237261 PMID: 1315042
Cannabinoids inhibit N-type calcium channels in neuroblastoma-glioma cells. May 1 1992

11/6/43 (Item 1 from file: 5) 12890636 BIOSIS NO.: 200100097785
Novel peptide analgesic from mollusc-hunting cone snail. 2000

11/6/44 (Item 2 from file: 5) 12889738 BIOSIS NO.: 200100096887
Novel dehydropyridine Ca entry blocker, cilnidipine, attenuates hyperalgesia in relation to apoptosis of spinal cord neurons in rat. 2000

11/6/45 (Item 3 from file: 5) 12889737 BIOSIS NO.: 200100096886
Role of spinal Ca channel in modulating inflammatory pain response in rats. 2000

11/6/46 (Item 4 from file: 5) 12881219 BIOSIS NO.: 200100088368
Neuropathic injury reduces T-type calcium current but not R-type in rats. 2000

11/6/47 (Item 5 from file: 5) 12640268 BIOSIS NO.: 200000393770
Synthesis and biological activity of 4-aminopiperidine derivatives as N-type calcium channel antagonists. 2000

11/6/48 (Item 6 from file: 5) 12339591 BIOSIS NO.: 200000093093
ProsaptideTM D5 reverses hyperalgesia: Inhibition of calcium channels through a pertussis toxin-sensitive G-protein mechanism in the rat. 2000

11/6/49 (Item 7 from file: 5) 12049064 BIOSIS NO.: 199900329583
Myenteric release of acetylcholine is impaired in ileal but not in colonic inflammation : Ca2+ channel sub-type dependence. 1999

11/6/50 (Item 8 from file: 5) 12026515 BIOSIS NO.: 199900307034
Methods and formulations for preventing progression of neuropathic pain . 1999

11/6/51 (Item 9 from file: 5) 12015412 BIOSIS NO.: 199900295931
Polypeptide omega - conotoxin GVIA as a basis for new analgesic and neuroprotective agents. 1999

11/6/52 (Item 10 from file: 5) 11834638 BIOSIS NO.: 199900080747
Blockade of spinal calcium channels does not potentiate the antinociceptive effect of morphine: An electrophysiological study. 1998

11/6/53 (Item 11 from file: 5) 11315844 BIOSIS NO.: 199800097176
Calcium channel blockers suppress the responses of rat dorsal horn cell to nociceptive input. 1997

11/6/54 (Item 12 from file: 5) 10838612 BIOSIS NO.: 199799459757
Blockade of spinal N- and P-type, but not L-type, calcium channels inhibits the excitability of rat doral horn neurones produced by subcutaneous formalin inflammation . 1997

11/6/55 (Item 13 from file: 5) 10197321 BIOSIS NO.: 199698652239
Mechanism of prostaglandin E-2-induced substance P release from cultured sensory neurons. 1996

11/6/56 (Item 14 from file: 5) 08990795 BIOSIS NO.: 199396142296
Regulation of neuropeptide release from pulmonary capsaicin-sensitive afferents in relation to bronchoconstriction. 1993

11/7/3 (Item 3 from file: 155) DIALOG(R)File 155:MEDLINE(R)
11788623 21320133 PMID: 11426839
Antinociceptive action of amlodipine blocking N-type Ca2+ channels at the primary afferent neurons in mice.
Murakami M; Nakagawasai O; Fujii S; Kameyama K; Murakami S; Hozumi S; Esashi A; Taniguchi R; Yanagisawa T; Tan-no K; Tadano T; Kitamura K; Kisara K
Department of Molecular Pharmacology, Tohoku University School of Medicine, Sendai, Japan. mmura@mail.cc.tohoku.ac.jp
European journal of pharmacology (Netherlands) May 11 2001, 419 (2-3) p175-81, ISSN 0014-2999 Journal Code: EN6 Languages: ENGLISH Document type: Journal Article Record type: Completed
We investigated the antinociceptive action of amlodipine, a dihydropyridine derivative, which acts on both L- and N-type voltage-dependent Ca2+ channels (VDCCs), in mice. Intrathecal injection of amlodipine (300 nmol/kg) significantly shortened the licking time in the late phase of a formalin test, while no effect was found with another dihydropyridine derivative, nicardipine (300 nmol/kg). Cilnidipine and omega - conotoxin GVIA also showed marked analgesic effects under the same experimental conditions. Transcripts of alpha1A, alpha1B, alpha1E, alpha1F, alpha1H, beta3, and beta4 subunits were detected by polymerase-chain reaction (PCR) in the dorsal root ganglion, suggesting the existence of a variety of voltage-dependent Ca2+ channels . Electrophysiological experiments showed that amlodipine and cilnidipine inhibit N-type currents in the dorsal root ganglion cells. These results suggest that amlodipine, cilnidipine, and omega - conotoxin GVIA exert their antinociceptive actions by blocking N-type Ca2+ channels in the primary nociceptive afferent fibers. Blocking of the Ca2+ channels results in attenuation of synaptic transmission of nociceptive neurons. Furthermore, it is suggested that some N-type Ca2+ channel blockers might have therapeutic potential as analgesics when applied directly into the subarachnoidal space. Record Date Created: 20010621

11/7/5 (Item 5 from file: 155) DIALOG(R)File 155:MEDLINE(R)
11661155 21387571 PMID: 11496122
Differential nociceptive responses in mice lacking the alpha(1B) subunit of N-type Ca(2+) channels .
Hatakeyama S; Wakamori M; Ino M; Miyamoto N; Takahashi E; Yoshinaga T; Sawada K; Imoto K; Tanaka I; Yoshizawa T; Nishizawa Y; Mori Y; Niidome T; Shoji S
Department of Neurology, Institute of Clinical Medicine, University of Tsukuba, Tsukuba, Ibaraki 305-8575, Japan.
Neuroreport (England) Aug 8 2001, 12 (11) p2423-7, ISSN 0959-4965 Journal Code: A6M Languages: ENGLISH Document type: Journal Article Record type: Completed
The role of N-type Ca(2+) channels in nociceptive transmission was examined in genetically engineered mice lacking the alpha(1B) subunit of N-type channels and in their heterozygote and wild-type littermates. In alpha(1B)-deficient mice, N-type channel activities in dorsal root ganglion neurons and spinal synaptoneurosomes were eliminated without compensation by other types of voltage-dependent Ca(2+) channels . The alpha(1B)-deficient mice showed a diminution in the phase 2 nociceptive responses more extensively than in the phase 1 nociceptive responses of the formalin test. The alpha(1B)-deficient mice exhibited significantly increased thermal nociceptive thresholds in the hot plate test, but failed to increase mechanical nociceptive thresholds in the tail pinch test. These results suggest a crucial role of N-type channels in nociceptive transmission, especially for persistent pain like phase 2 of the formalin test and for nociception induced by thermal stimuli. Record Date Created: 20010809

11/7/8 (Item 8 from file: 155) DIALOG(R)File 155:MEDLINE(R)
11552881 21276249 PMID: 11382408
Distribution of various calcium channel alpha(1) subunits in murine DRG neurons and antinociceptive effect of omega - conotoxin SVIB in mice.
Murakami M; Suzuki T; Nakagawasai O; Murakami H; Murakami S; Esashi A; Taniguchi R; Yanagisawa T; Tan-No K; Miyoshi I; Sasano H; Tadano T
Department of Molecular Pharmacology, Tohoku University School of Medicine, Seiryoumachi, Aoba-ku, Sendai 980-8575, Japan. mmura@mail.cc.tohoku.ac.jp
Brain research (Netherlands) Jun 8 2001, 903 (1-2) p231-6, ISSN 0006-8993 Journal Code: B5L Languages: ENGLISH Document type: Journal Article Record type: Completed
Immunohistological study revealed the differential localization of subtypes of voltage-dependent calcium channels in the dorsal root ganglion neurons. Intrathecal injection of omega - conotoxin SVIB, an analogue of omega - conotoxin GVIA, which acts on N-type voltage-dependent calcium channels , significantly shortened the licking time in the late phase of a formalin test. Record Date Created: 20010530

11/7/11 (Item 11 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10914159 20578057 PMID: 11133635
The combined effects of N-type calcium channel blockers and morphine on A delta versus C fiber mediated nociception.
Pirec V; Laurito CE; Lu Y; Yeomans DC
Department of Psychiatry, University of Illinois at Chicago College of Medicine, Chicago, Illinois, USA.

Anesthesia and analgesia (UNITED STATES) Jan 2001, 92 (1) p239-43, ISSN 0003-2999 Journal Code: 4R8
Contract/Grant No.: DA08256, DA, NIDA Languages: ENGLISH Document type: Journal Article Record type: Completed
Intrathecal mu opiates produce analgesia presynaptically by inhibiting calcium ion influx and postsynaptically by increasing potassium flux. Mu receptors are expressed on presynaptic terminals of unmyelinated (C), but not myelinated (A delta) nociceptors. Thus, mu-opioids such as morphine may act presynaptically to inhibit C, but not A delta, neurotransmission, and postsynaptically on dorsal horn cells that receive input from A delta and/or C fiber nociceptors. N-type calcium ion channel blockers, such as omega - conotoxin GVIA (omega -CTX), produce analgesia by impeding flux of calcium ions into A delta and C fiber nociceptor terminals. Thus, morphine and omega -CTX attenuated C fiber nociception additively, possibly indicating the same presynaptic site of action. Conversely, morphine and omega - CTX were supraadditively analgesic on an A delta test, indicating that these agents probably have different sites of action. We conclude that although intrathecal application of either morphine or omega -CTX attenuates both A delta and C fiber mediated nociception in rats, the combined effects are quite different for the two fiber types. Specifically, although coadministration of morphine with omega -CTX produces an additive, apparently presynaptic antinociception for C fiber-mediated responses, the combination produces a clearly supraadditive, and likely synergistic effect on A delta mediated nociception, probably by acting at pre and postsynaptic sites, respectively. Implications: This study demonstrates that combined spinal administration of mu opioids and N-type calcium channel blockers may be useful in providing analgesia for A delta mediated (first, sharp) pain while minimizing the side effects of both drugs. Record Date Created: 20010109

11/7/13 (Item 13 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10663729 20283152 PMID: 10822250
Structure-activity relationships of omega -conotoxins at N-type voltage-sensitive calcium channels .
Nielsen KJ; Schroeder T; Lewis R
Centre for Drug Design and Development (3D), Institute for Molecular Bioscience, The University of Queensland, Brisbane, Australia.
Journal of molecular recognition (ENGLAND) Mar-Apr 2000, 13 (2) p55-70, ISSN 0952-3499 Journal Code: AO0
Languages: ENGLISH Document type: Journal Article; Review; Review, Tutorial Record type: Completed
Due to their selectivity towards voltage-sensitive calcium channels (VSCCs) omega -conotoxins are being exploited as a new class of therapeutics in pain management and may also have potential application in ischaemic brain injury. Here, the structure-activity relationships (SARs) of several omega -conotoxins including GVIA, MVIIA, CVID and MVIIC are explored. In addition, the three-dimensional structures of these omega -conotoxins and some structurally related peptides that form the cysteine knot are compared, and the effects of the solution environment on structure discussed. The diversity of binding and functional assays used to measure omega - conotoxin potencies at the N-type VSCC warranted a re-evaluation of the relationship between these assays. With one exception, [A22]-GVIA, this analysis revealed a linear correlation between functional (peripheral N-type VSCCs) and radioligand binding assays (central N-type VSCCs) for the omega -conotoxins and analogues that were tested over three studies. The binding and functional results of several studies are compared in an attempt to identify and distinguish those residues that are important in omega - conotoxin function as opposed to those that form part of the structural scaffold. Further to determining what omega - conotoxin residues are important for VSCC binding, the range of possible interactions between the ligand and channel are considered and the factors that influence the selectivity of MVIIA, GVIA and CVID towards N-type VSCCs examined. (70 Refs.) Record Date Created: 20000731

11/7/14 (Item 14 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10580919 20176956 PMID: 10714496
Design and biological evaluation of non-peptide analogues of omega - conotoxin MVIIA.
Menzler S; Bikker JA; Suman-Chauhan N; Horwell DC
Parke-Davis Neuroscience Research Centre, Cambridge, UK.
Bioorganic & medicinal chemistry letters (ENGLAND) Feb 21 2000, 10 (4) p345-7, ISSN 0960-894X Journal Code: C8B
Languages: ENGLISH Document type: Journal Article Record type: Completed
Omega - conotoxin MVIIA, a highly potent antagonist of the N-type voltage sensitive calcium channel , has shown utility in several models of pain and ischemia. We report a series of three alkylphenyl ether based analogues which mimic three key amino acids of the toxin. Two of the compounds have been found to exhibit IC50 values of 2.7 and 3.3 microM at the human N-type voltage sensitive calcium channel . Record Date Created: 20000515

11/7/22 (Item 22 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10309071 98290368 PMID: 9628404
Spinal application of omega - conotoxin GVIA, an N-type calcium channel antagonist, attenuates enhancement of dorsal spinal neuronal responses caused by intra-articular injection of mustard oil in the rat.
Nebe J; Vanegas H; Schaible HG
Physiologisches Institut der Universitat Wurzburg, Germany.
Experimental brain research. Experimentelle Himforschung. Experimentation cerebrale (GERMANY) May 1998, 120 (1) p61-9, ISSN 0014-4819 Journal Code: EP2 Languages: ENGLISH Document type: Journal Article Record type: Completed
Administration of the N-type calcium channel antagonist omega - conotoxin GVIA to the spinal cord reduces spinal neuronal responses to innocuous and noxious pressure applied to the knee, both in rats with normal knees and in rats in which a knee inflammation has induced a state of hyperexcitability in spinal neurons (Neugebauer et al. 1996, J Neurophysiol 76: 3740-3749). In the present experiments we studied whether the development of hyperexcitability of spinal neurons induced by intra-articular injection of mustard oil, an excitant of C-fibres, can be influenced by spinal pretreatment with omega - conotoxin GVIA. In anaesthetized rats, responses of wide-dynamic-range neurons were recorded in the spinal dorsal horn when standardized stimulation with innocuous and noxious pressure was applied to the knee and ankle joints. Injection of mustard oil into the knee joint cavity caused an initial neuronal discharge followed by an early (peaking at about 15 min) and a late (after 60 min) facilitation of responses to innocuous and noxious stimulation of the knee. Responses to ankle stimulation showed only the late facilitation. When omega - conotoxin GVIA (20 microl, 1 microM) was applied into a small trough onto the spinal cord above the recording site the responses to articular stimulation were reduced. Furthermore, when mustard oil was injected while omega - conotoxin GVIA was on the spinal cord, the early increase in the neuronal responses to innocuous pressure on the knee and the late increase in responses to noxious pressure on the ankle were significantly smaller than those observed in rats not treated with omega - conotoxin GVIA; the drop in the responses to noxious pressure on the knee was not significant. Thus the spinal application of omega - conotoxin GVIA reduced but did not completely prevent the fast and slow development of neuronal hyperexcitability of spinal cord neurons produced by a prompt and strong excitation of afferent C-fibres. This suggests that N-type calcium channels are important for the development of spinal cord hyperexcitability. Record Date Created: 19980827

11/7/24 (Item 24 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10295869 98081532 PMID: 9421179
Omega -agatoxin IVA, a P-type calcium channel antagonist, reduces nociceptive processing in spinal cord neurons with input from the inflamed but not from the normal knee joint--an electrophysiological study in the rat in vivo.
Nebe J; Vanegas H; Neugebauer V; Schaible HG
Physiologisches Institut der Universitat Wurzburg, Germany.
European journal of neuroscience (ENGLAND) Oct 1997, 9 (10) p2193-201, ISSN 0953-816X Journal Code: BY
Languages: ENGLISH Document type: Journal Article Record type: Completed
High threshold voltage-dependent P- and Q-type calcium channels are involved in neurotransmitter release. In order to investigate the role of P- and Q-type calcium channels in the mechanosensory (nociceptive) processing in the spinal cord, their participation in the responses of spinal wide-dynamic-range neurons to innocuous and noxious mechanical stimulation of the knee and ankle joints was studied in 30 anaesthetized rats. The knee was either normal or acutely inflamed by kaolin/carrageenan. During the topical application of omega -agatoxin IVA (P-type channel antagonist, 0.1 microM) onto the dorsal surface of the spinal cord, the responses to innocuous and noxious pressure applied to the normal knee were increased to respectively 124 +/- 42% and 114 +/- 23% of predrug values (mean +/- SD, $P < 0.05$, 14 neurons). By contrast, in rats with an inflamed knee, the responses to innocuous and noxious pressure applied to the knee were reduced to respectively 72 +/- 19 and 73 +/- 22% of baseline (mean +/- SD, $P < 0.01$, 13 neurons). In the same neurons, omega -agatoxin IVA slightly increased the responses to pressure on the non-inflamed ankle whether the knee was normal or inflamed. Thus P-type calcium channels seem to acquire a predominant importance in the excitation of spinal cord neurons by mechanosensory input from inflamed tissue and hence in the generation of inflammatory pain . By contrast, the Q-type channel antagonist, omega - conotoxin MVIIC (1 or 100 microM), had no significant effect upon responses to innocuous or noxious pressure applied to either normal or inflamed knees (25 neurons). Record Date Created: 19980204

11/7/25 (Item 25 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10290745 97404278 PMID: 9262364
Differential effects of omega - conotoxin GVIA, nimodipine, calmidazolium and KN-62 injected intrathecally on the antinociception induced by beta-endorphin, morphine and [D-Ala2,N-MePhe4,Gly-ol5]-enkephali n administered intracerebroventricularly in the mouse.
Suh HW; Song DK; Choi SR; Huh SO; Kim YH
Department of Pharmacology, Institute of Natural Medicine, College of Medicine, Hallym University, Chunchon, Kangwon-Do, South Korea. hwsuh@sun.hallym.ac.kr
Journal of pharmacology and experimental therapeutics (UNITED STATES) Aug 1997, 282 (2) p961-6, ISSN 0022-3565 Journal Code: JP3 Languages: ENGLISH Document type: Journal Article Record type: Completed
We previously reported that beta-endorphin and morphine administered supraspinally produce antinociception by activating different descending pain -inhibitory systems. To determine the role of spinal calcium channels , calmodulin and calcium/calmodulin-dependent protein kinase II in the production of antinociception induced by morphine, [D-Ala2,N-MePhe4,Gly-ol5]-enkephalin (DAMGO) or beta-endorphin administered supraspinally, the effects of nimodipine (an L-type calcium channel blocker), omega - conotoxin GVIA (an N-type voltage-dependent calcium channel blocker), calmidazolium (a calmodulin antagonist) or KN-62 (a calcium/calmodulin-dependent protein kinase II inhibitor) injected intrathecally (i.t.) on the antinociception induced by morphine, DAMGO or beta-endorphin administered intracerebroventricularly (i.c.v.) were examined in the present study. Antinociception was assessed by the mouse tail-flick test. The i.t. injection of nimodipine (from 0.024 to 2.4 pmol), omega - conotoxin GVIA (from 0.0033 to 0.33 pmol), calmidazolium (from 0.0015 to 0.15 pmol) or KN-62 (from 0.0014 to 0.14 pmol) alone did not affect the basal tail-flick latencies. The i.t. pretreatment of mice with nimodipine, omega - conotoxin GVIA, calmidazolium or KN-62 dose dependently attenuated the inhibition of the tail-flick response induced by beta-endorphin administered i.c.v. However, the inhibition of the tail-flick response induced by morphine or DAMGO administered i.c.v. was not changed by i.t. pretreatment with nimodipine, omega - conotoxin GVIA, calmidazolium or KN-62. The results suggest that

spinally located L- and N-type calcium channels , calmodulin and calcium/calmodulin-dependent protein kinase II may be involved in the modulation of antinociception induced by beta-endorphin, but not morphine and DAMGO, administered supraspinally. Record Date Created: 19970911

11/7/27 (Item 27 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10109296 98398342 PMID: 9729273
Effect of subcutaneous administration of calcium channel blockers on nerve injury-induced hyperalgesia.
White DM; Cousins MJ
Department of Anaesthesia and Pain Management, Royal North Shore Hospital, St. Leonards, N.S.W., 2065, Australia. dmwhite@med.usyd.edu.au
Brain research (NETHERLANDS) Aug 10 1998, 801 (1-2) p50-8, ISSN 0006-8993 Journal Code: B5L Languages: ENGLISH Document type: Journal Article Record type: Completed
Recent studies suggest that calcium contributes to peripheral neural mechanisms of hyperalgesia associated with nerve damage. In this animal behavioural study, we examined further the contribution of calcium in neuropathic pain by testing whether subcutaneous administration of either a calcium chelating agent or voltage-dependent calcium channel blockers attenuate nerve injury-induced hyperalgesia to mechanical stimulation. Studies were carried out in animals with partially ligated sciatic nerves, an established animal model of neuropathic pain . The nociceptive flexion reflex was quantified using an Ugo Basile Analgesymeter. Partial nerve injury induced a significant decrease in mechanical threshold compared to the sham operated controls. Daily subcutaneous injections of the calcium chelating agent, Quin 2 (20 microgram/2.5 microliter), significantly attenuated the nerve injury-induced hyperalgesia. Similarly, SNX-111, a N-type channel blocker, also significantly attenuated the nerve injury-induced hyperalgesia. SNX-230, a P and/or Q-type channel blocker, and nifedipine, a L-type channel blocker, had no effect on the hyperalgesia to mechanical stimulation. In control experiments, SNX-111 had no effect on mechanical thresholds when administered subcutaneously in either the hindpaw of normal animals or the back of the neck in nerve injury animals. This study shows that neuropathic pain involves a local calcium-dependent mechanism in the receptive field of intact neurons of an injured nerve, since it can be alleviated by subcutaneous injections of either a calcium chelating agent or SNX-111, a N-type calcium channel blocker. These agents may be effective, peripherally acting therapeutic agents for neuropathic pain . Copyright 1998 Elsevier Science B.V. Record Date Created: 19990512

11/7/28 (Item 28 from file: 155) DIALOG(R)File 155:MEDLINE(R)
09983819 99006668 PMID: 9792182
Pharmacotherapeutic potential of omega - conotoxin MVIIA (SNX-111), an N-type neuronal calcium channel blocker found in the venom of Conus magus.
Bowersox SS; Luther R
Department of Pharmacology, Neurex Corporation, Menlo Park, CA 94025, USA.
Toxicon (ENGLAND) Nov 1998, 36 (11) p1651-8, ISSN 0041-0101 Journal Code: VWT Languages: ENGLISH Document type: Journal Article; Review; Review, Tutorial Record type: Completed (32 Refs.) Record Date Created: 19990114

11/7/35 (Item 35 from file: 155) DIALOG(R)File 155:MEDLINE(R)
09222381 96416732 PMID: 8819527
Spinal morphine/clonidine antinociceptive synergism: involvement of G proteins and N-type voltage-dependent calcium channels
Wei ZY; Karim F; Roerig SC
Department of Pharmacology, Louisiana State University Medical Center, Shreveport, USA.
Journal of pharmacology and experimental therapeutics (UNITED STATES) Sep 1996, 278 (3) p1392-407, ISSN 0022-3565 Journal Code: JP3 Contract/Grant No.: DA07972, DA, NIDA Languages: ENGLISH Document type: Journal Article Record type: Completed
When morphine and clonidine are coadministered into the spinal cord (intrathecally) the resulting antinociception is greater than would be expected if the drug responses were additive; thus, a synergistic interaction. The mechanism for this synergistic interaction was investigated using agents which alter calcium channel function and G protein function. Drugs were administered intrathecally to mice and antinociception was measured using the tail flick test. The L-type calcium channel antagonists nifedipine (15 micrograms) and verapamil (15 micrograms) and the N-type antagonist omega - conotoxin GVIA (3 and 30 ng) decreased ED50 values for both morphine and clonidine three-to five-fold. The L-type calcium channel activator Bay K 8644 had a biphasic effect; 1.7 ng increased, although 170 ng decreased, morphine and clonidine ED50 values. None of the calcium channel modifiers affected the morphine/clonidine synergism. In mice pretreated with pertussis toxin (PTX, one, 10-ng dose 21 days previously), the morphine ED50 value increased two-fold, although the clonidine ED50 value was not changed. PTX pretreatment did not alter the morphine/clonidine synergism. Also, in PTX-pretreated mice, nifedipine and 1.7 ng Bay K 8644 did not alter the morphine/clonidine synergism. However, in PTX-pretreated animals omega - conotoxin GVIA (3 ng) changed the morphine/clonidine synergism to an additive interaction. Thus, both N-type calcium channels and PTX-sensitive G proteins are likely involved in spinal morphine/clonidine synergism. Record Date Created: 19961105

11/7/37 (Item 37 from file: 155) DIALOG(R)File 155:MEDLINE(R)
08899552 95363679 PMID: 7636726
Synthetic omega -conopeptides applied to the site of nerve injury suppress neuropathic pains in rats.
Xiao WH; Bennett GJ
Neurobiology and Anesthesiology Branch, National Institute of Dental Research, National Institutes of Health, Bethesda, Maryland, USA.
Journal of pharmacology and experimental therapeutics (UNITED STATES) Aug 1995, 274 (2) p666-72, ISSN 0022-3565 Journal Code: JP3 Languages: ENGLISH Document type: Journal Article Record type: Completed
In patients and animals with painful peripheral neuropathies, spontaneous ectopic discharge from injured primary afferents is hypothesized to maintain a central state of hyperexcitability that underlies hyperalgesia and allodynia. Temporary suppression of this discharge allows the central state to normalize, such that hyperalgesia and allodynia are absent or reduced until the resumption of the discharge rekindles central hyperexcitability. Previous work suggests that Ca++ channels are involved in the genesis of spontaneous discharge from injured afferents. We applied SNX-111 and SNX-124 (0.1-3.0 micrograms), synthetic homologs of omega -conopeptides (MVIIA and GVIA, respectively) and potent blockers of neuronal N-type voltage-sensitive Ca++ channels , to the site of nerve injury via chronically implanted perineural cannulae in rats with an experimental painful peripheral neuropathy (the chronic constriction injury model). Heat-hyperalgesia and mechano-allodynia were reduced for at least 3 hr. Drug application to a normal nerve had no effect on responses to heat or mechanical stimuli. These results suggest that N-type Ca++ channel blockers may be useful in the treatment of the abnormal pains that occur after nerve injury. Record Date Created: 19950908

11/7/43 (Item 1 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2002 BIOSIS. All rts. reserv.
12890636 BIOSIS NO.: 200100097785
Novel peptide analgesic from mollusc-hunting cone snail.
AUTHOR: McIntosh J M(a); Corpuz G O; Layer R T; Garrett J E; Wagstaff J D; Vyazovkina A; Bulaj G; Cruz L J; Olivera B M
AUTHOR ADDRESS: (a)University of Utah, Salt Lake City, UT**USA
JOURNAL: Society for Neuroscience Abstracts 26 (1-2):pAbstract No-4004 2000
MEDIUM: print CONFERENCE/MEETING: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 SPONSOR: Society for Neuroscience ISSN: 0190-5295 RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
ABSTRACT: Cone snails are tropical marine molluscs that envenomate their prey with a complex mixture of pharmacologically active compounds. Due to their high potency and selectivity, several cone snail-derived peptides are under development for the treatment of human disorders. Specific examples are omega - conotoxin MVIIA (ziconotide), an N-type calcium channel antagonist, and contulakin-G, a neurotensin agonist. Both peptides, isolated from fish-hunting cone snails, show promise as novel agents for treatment of pain syndromes. We now report the purification and biochemical characterization of a novel twelve amino acid, disulfide-rich conopeptide from a mollusc-hunting cone snail that produces dose-dependent analgesia in mice as measured by a hot-plate test. This peptide is structurally unrelated to previously isolated conotoxins. Intrathecal doses (0.1 nmol-10 nmol) that produce analgesia do not produce motor impairment as measured by rotorod test. Thus, the new cone venom peptide represents a novel lead for conopeptide analgesics.

11/7/55 (Item 13 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2002 BIOSIS. All rts. reserv.
10197321 BIOSIS NO.: 199698652239
Mechanism of prostaglandin E-2-induced substance P release from cultured sensory neurons.
AUTHOR: White D M
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JOURNAL: Neuroscience 70 (2):p561-565 1996 ISSN: 0306-4522 DOCUMENT TYPE: Article RECORD TYPE: Abstract
LANGUAGE: English
ABSTRACT: Hyperalgesia (tenderness) is a prominent feature of the inflammatory response. It is thought to be mediated, in part, by humoral factors such as prostaglandin E-2, which act directly to sensitize primary afferent nociceptors. Prostaglandin E-2 also interacts with nociceptors to induce a release of substance P, which can feed back to enhance the inflammatory response and also induce a long-lasting hyperalgesia. This study examined the mechanism of prostaglandin E-2-induced substance P release from cultured adult rat dorsal root ganglion cells. Release studies were performed by bathing cultures with Tyrode solution +- test agents and substance P was measured by radioimmunoassay. Substance P release induced by 100 nM prostaglandin E-2 was inhibited by the prostaglandin antagonist, SC19220, and modulated by the guanine nucleotide analogs, guanosine-5'-(gamma-thio)triphosphate and guanosine-5'-(beta-thio)diphosphate, which stimulate and inhibit, respectively, stimulatory G-proteins. Substance P release was found to be Ca-2+-dependent, requiring an influx of Ca-2- via N-type voltage-sensitive Ca-2+ channels , since it was blocked by omega - conotoxin , but not nifedipine. The results suggest that prostaglandin E-2 acts via a G-protein-coupled binding site on dissociated dorsal root ganglion cells to induce a Ca-2+-dependent release of substance P, and provide further insight into the possible mechanisms underlying hyperalgesia associated with inflammation .

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Hypoxia -induced secretion of serotonin from intact pulmonary neuroepithelial bodies in neonatal rabbit. Mar 1 2002

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Myocardial interstitial norepinephrine and dihydroxyphenylglycol levels during ischemia and reperfusion. Jan 2001

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Autoradiographic localization of N-type VGCCs in gerbil hippocampus and failure of omega - conotoxin MVIIA to attenuate neuronal injury after transient cerebral ischemia . Jul 13 2001

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Involvement of Na+ and Ca2+ channel activation and resultant nitric oxide synthesis in glutamate-mediated hypoxic injury in rat cerebrocortical slices. Sep 29 2000

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Differential acetylcholine release mechanisms in the ischemic and non-ischemic myocardium. Mar 2000

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Adenosine inhibits L-type Ca2+ current and catecholamine release in the rabbit carotid body chemoreceptor cells. Feb 1999

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The role of voltage-gated Ca2+ channels in anoxic injury of spinal cord white matter. Jan 30 1999

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Hypoxia evokes catecholamine secretion from rat pheochromocytoma PC-12 cells. Jul 9 1998

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Alterations in K+ evoked profiles of neurotransmitter and neuromodulator amino acids after focal ischemia -reperfusion. Mar 1998

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SNX-111, a novel, presynaptic N-type calcium channel antagonist, is neuroprotective against focal cerebral ischemia in rabbits. Dec 9 1997

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A novel Na+/Ca2+ channel blocker, NS-7, suppresses hypoxic injury in rat cerebrocortical slices. Aug 1998

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Intracellular inositol 1,3,4,5-tetrakisphosphate enhances the calcium current in hippocampal CA1 neurones of the gerbil after ischaemia. Nov 15 1996

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Effects of Ca2+ and Na+ channel inhibitors in vitro and in global cerebral ischaemia in vivo. Aug 6 1997

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Selective changes in cell bodies and growth cones of nerve growth factor-differentiated PC12 cells induced by chemical hypoxia . Aug 1997

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Hypoxia -induced catecholamine release and intracellular Ca2+ increase via suppression of K+ channels in cultured rat adrenal chromaffin cells. Jul 1997

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Role of Ca2+ in metabolic inhibition-induced norepinephrine release in rat brain synaptosomes. Feb 1997

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Effects of fructose-1,6-bisphosphate on glutamate release and ATP loss from rat brain slices during hypoxia . Oct 1996

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Characterization of a chemical anoxia model in cerebellar granule neurons using sodium azide: protection by nifedipine and MK-801. Apr 1 1996

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Decreased calcium accumulation in isolated nerve endings during hibernation in ground squirrels. Aug 1996

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Selective N-type calcium channel antagonist omega conotoxin MVIIA is neuroprotective against hypoxic neurodegeneration in organotypic hippocampal-slice cultures. Nov 1996

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Transient brain ischemia in rabbits: the effect of omega -conopeptide MVIIC on hippocampal excitatory amino acids. Sep 18 1995

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Voltage-gated calcium channels in CNS white matter: role in anoxic injury. Jul 1995

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Ca2+-dependent and -independent mechanisms of ischaemia-evoked release of [3H]-dopamine from rat striatal slices. Apr 1995

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Long-term modulation of inward currents in O2 chemoreceptors by chronic hypoxia and cyclic AMP in vitro. Mar 1995

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Omega - conotoxin GVIA protects against ischemia -induced neuronal death in the Mongolian gerbil but not against quinolinic acid-induced neurotoxicity in the rat. Feb 1994

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Pharmacological profile of a novel neuronal calcium channel blocker includes reduced cerebral damage and neurological deficits in rat focal ischemia . May 1994

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Functional identification of histamine H3-receptors in the human heart. Jul 1995

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Pre- and post-synaptic modulators of excitatory neurotransmission: comparative effects on hypoxia /hypoglycemia in cortical cultures. Apr 18 1994

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Autoradiographic analysis of L- and N-type voltage-dependent calcium channel binding in canine brain after global cerebral ischemia /reperfusion. Sep 19 1994

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Effects of calcium antagonists on hypoxic and NMDA injury in rat hippocampal slices. 1994

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Solution structure of omega - conotoxin GVIA using 2-D NMR spectroscopy and relaxation matrix analysis. Jul 27 1993

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A selective N-type calcium channel antagonist protects against neuronal loss after global cerebral ischemia . Aug 15 1993

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Inhibition of ischemia -induced dopamine release by omega - conotoxin , a calcium channel blocker, in the striatum of spontaneously hypertensive rats: in vivo brain dialysis study. Jan 1992

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Treatment with conotoxin , an 'N-type' calcium channel blocker, in neuronal hypoxic-ischemic injury. Dec 24 1990

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Flunarizine blocks elevation of free cytosolic calcium in synaptosomes following sustained depolarization. Nov 1993

14/6/64 (Item 1 from file: 5) 13382490 BIOSIS NO.: 200200011311
Effects of ketamine on in vivo cardiac sympathetic nerve endings. 2001

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Characteristics of protective effects of NMDA antagonist and calcium channel antagonist on ischemic calcium accumulation in rat hippocampal CA1 region. 2001

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Effects of ion channel blockade on the distribution of Na, K, Ca and other elements in oxygen-glucose deprived CA1 hippocampal neurons. 2001

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Synthesis and biological activity of 4-aminopiperidine derivatives as N-type calcium channel antagonists. 2000

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Effects of omega -agatoxin and omega - conotoxin GVIA on ischaemia-induced dopamine release in vitro. 1996

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Effects of omega - conotoxin MVIIC on veratridine-induced cytotoxicity and cytosolic Ca-2+ oscillations. 1996

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Functional identification of histamine H-3-receptors in the human heart. 1995

14/6/71 (Item 8 from file: 5) 08916320 BIOSIS NO.: 199396067821
Role of neuronal and vascular calcium- channels in the ACTH-induced reversal of haemorrhagic shock. 1993

14/6/72 (Item 9 from file: 5) 08724105 BIOSIS NO.: 199395013456
Potentiation of potassium-evoked noradrenaline and neuropeptide Y co-release by cardiac energy depletion: Role of calcium channels and sodium-proton exchange. 1992

14/7/4 (Item 4 from file: 155) DIALOG(R)File 155:MEDLINE(R) 11703969 21324260 PMID: 11430886
Autoradiographic localization of N-type VGCCs in gerbil hippocampus and failure of omega - conotoxin MVIIA to attenuate neuronal injury after transient cerebral ischemia .
Azimi-Zonooz A; Kawa CB; Dowell CD; Olivera BM
Department of Pediatrics, Oregon Health Sciences University, Portland, OR 97201, USA. azimi@biology.utah.edu
Brain research (Netherlands) Jul 13 2001, 907 (1-2) p61-70, ISSN 0006-8993 Journal Code: B5L Contract/Grant No.: GM48677, GM, NIGMS; K12-HD00850, HD, NICHD Languages: ENGLISH Document type: Journal Article Record type: Completed
In the mammalian central nervous system, transient global ischemia of specific duration causes selective degeneration of CA1 pyramidal neurons in hippocampus. Many of the ischemia -induced pathophysiologic cascades that destroy the neurons are triggered by pre- and postsynaptic calcium entry. Consistent with this, many calcium channel blockers have been shown to be neuroprotective in global models of ischemia . omega - Conotoxin MVIIA, a selective N-type VGCC blocker isolated from the venom of Conus magus, protects CA1 neurons in the rat model of global ischemia , albeit transiently. The mechanism by wh[redacted] this peptide renders neuroprotection is unknown. We performed high-resolution receptor autoradiography with the radiolabel[redacted] peptide and observed highest binding in stratum lucidum of CA3 subfield, known to contain inhibitory neurons potentially important in the pathogenesis of delayed neuronal death. This finding suggested that the survival of stratum lucidum inhibitory neurons might be the primary event, leading to CA1 neuroprotection after ischemia . Testing of this hypothesis required the reproduction of its neuroprotective effects in the gerbil model of global ischemia . Surprisingly, we found that omega -MVIIA did not attenuate CA1 hippocampal injury after 5 min of cerebral ischemia in gerbil. Possible reasons are discussed. Lastly, we show that the peptide can be used as a synaptic marker in assessing short and long-term changes that occur in hippocampus after ischemic injury. Record Date Created: 20010629

14/7/5 (Item 5 from file: 155) DIALOG(R)File 155:MEDLINE(R)
11278455 21095684 PMID: 11172784
Release of substance P by low oxygen in the rabbit carotid body: evidence for the involvement of calcium channels .
Kim DK; Oh EK; Summers BA; Prabhakar NR; Kumar GK
Department of Biochemistry, School of Medicine, Case Western Reserve University, Cleveland, OH 44106, USA.
Brain research (Netherlands) Feb 23 2001, 892 (2) p359-69, ISSN 0006-8993 Journal Code: B5L
Contract/Grant No.: HL-25830, HL, NHLBI; HL-46462, HL, NHLBI Languages: ENGLISH
Document type: Journal Article Record type: Completed
Carotid bodies from diverse species contain substance P (SP), an 11-residue peptide that belongs to the tachykinin peptide family. Previous studies indicated that SP is excitatory to the carotid body and is associated with sensory response to hypoxia . However, release of SP from the carotid body during hypoxia has not been documented. In the present study, we determined whether hypoxia releases SP from the carotid body and further characterized the mechanism(s) associated with SP release by low oxygen. The release of SP from superfused rabbit carotid body was determined by an enzyme immunoassay (EIA). SP-[redacted] immunoreactivity was localized to many glomus cells and nerve fibers and the concentration of SP in the rabbit carotid body [redacted] 1.5+/-0.1 ng/mg protein. For release studies, carotid bodies (n=56) were superfused with a modified Tyrode medium containing Hepes buffer, pH 7.4, saturated with either room air (normoxia) or hypoxic gas mixtures. The basal release of SP during normoxia was 51.0+/-1.5 fmol/min per mg protein. Hypoxia increased SP release from the carotid body and the magnitude of release is dependent on the severity of hypoxic stimulus. Moderate hypoxia (pO2, 79+/-4 mmHg) stimulated SP release by approximately 50%, whereas SP release during severe hypoxia (pO2, 11+/-6 mmHg) was 2-fold higher than the normoxic control. A similar pattern of SP release was also observed when superfusion medium containing CO2-HCO3 buffer, pH 7.4, was used for release studies. To examine the mechanism(s) associated with hypoxia -induced SP release from the carotid body, moderate level of hypoxia (12% O2+N2) was used. Omission of calcium in the superfusion medium markedly attenuated hypoxia -induced SP release (>95%), whereas the basal release of SP was unaffected. Cd2+ (100 microM), a voltage-dependent Ca2+ channel blocker, abolished hypoxia -induced SP release. About 85% of SP release by hypoxia was inhibited by omega - conotoxin GVIA (1 microM), an N-type Ca2+ channel blocker, whereas nitrendipine (1.5 microM), an inhibitor of L-type Ca2+ channel partially attenuated (approximately 65%) hypoxia -induced SP release. By contrast, omega -agatoxin TK (50 nM), a P/Q-type Ca2+ channel inhibitor, had no significant effect (P>0.05, n=6). These results suggest that SP is released from the rabbit carotid body by hypoxia that depends on the severity of the hypoxic stimulus. Further, SP release by hypoxia is a calcium-dependent process and is primarily mediated by N- and L-type Ca2+ channels . Record Date Created: 20010222

14/7/6 (Item 6 from file: 155 DIALOG(R)File 155:MEDLINE(R)
10838000 20516993 PMID: 11065180
Involvement of Na+ and Ca2+ channel activation and resultant nitric oxide synthesis in glutamate-mediated hypoxic injury in rat cerebrocortical slices.
Oka M; Itoh Y; Ukai Y
Research Laboratories, Nippon Shinyaku Co., Ltd., Kyoto, Japan.
Life sciences (ENGLAND) Sep 29 2000, 67 (19) p2331-43, ISSN 0024-3205 Journal Code: L62 Languages: ENGLISH Document type: Journal Article Record type: Completed
The role of Na+ and Ca2+ channels in glutamate-mediated hypoxic injury was investigated in slices of the rat cerebral cortex. Hypoxic injury was determined by mitochondrial reduction of 3-(4,5-dimethyl-2-thiazol)-2,5-dip henyltetrazolium bromide after exposure of brain slices to 30-min of hypoxia /glucose deprivation followed by 3-h of reoxygenation. Endogenous glutamate release was markedly elevated during hypoxia /glucose deprivation, but it returned almost to basal level during reoxygenation. Hypoxic injury was prevented by MK-801 or 6-cyano-7-nitroquinoxaline-2,3-di one. Combined treatment with omega - conotoxin GVIA, omega -agatoxin IVA, and tetrodotoxin reversed the hypoxic injury, although none of these agents alone or nifedipine was effective. Moreover, a novel Na+/Ca2+ channel blocker NS-7 [4-(4-fluorophenyl)-2-methyl-6-(5-piperidinopentylox y) pyrimidine hydrochloride] significantly inhibited the hypoxic injury. Several inhibitors of nitric oxide synthase also blocked the hypoxic injury. Consistently, nitric oxide synthesis, as estimated from cyclic GMP formation in the extracellular fluids, was enhanced during hypoxia /glucose deprivation. NS-7 and other Na+ and Ca2+ channel blockers suppressed the enhancement of nitric oxide synthesis, although these compounds alone, or in combination, did not reduce hypoxic glutamate release. These findings suggest that hypoxic injury in rat cerebrocortical slices is triggered by glutamate and subsequent enhancement of nitric oxide synthesis through activation of both Na+ and Ca2+ channels . Thus, the simultaneous blockade of both Na+ channel as well as N-type and P/Q-type Ca2+ channels is required to sufficiently reverse the hypoxic injury. Record Date Created: 20001106

14/7/14 (Item 14 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10619229 20237760 PMID: 10773024
Preferential inhibition by a novel Na(+)/Ca(2+) channel blocker NS-7 of severe to mild hypoxic injury in rat cerebrocortical slices: A possible involvement of a highly voltage-dependent blockade of Ca(2+) channel .
Oka M; Itoh Y; Ukai Y
Research Laboratories, Nippon Shinyaku Co., Ltd., Nishiohji Hachijo Minami-ku, Kyoto, Japan. m.oka@po.nippon-shinyaku-co.jp
Journal of pharmacology and experimental therapeutics (UNITED STATES) May 2000, 293 (2) p522-9, ISSN 0022-3565 Journal Code: JP3 Languages: ENGLISH Document type: Journal Article Record type: Completed
The hypoxic injury was induced in rat cerebrocortical slices by the exposure to hypoxia for 45 min in the absence or presence of 3 mM glucose, followed by reoxygenation for 5 h. The injury was more pronounced in the absence of glucose (severe hypoxic injury) than in the presence of glucose (mild hypoxic injury). A novel Na(+)/Ca(2+) channel blocker, NS-7 [4-(4-fluorophenyl)-2-methyl-6-(5-piperidinopentyloxy) pyrimidine hydrochlo ride], at 3 to 30 microM inhibited preferentially the severe hypoxic injury, whereas MK-801, omega - conotoxin GVIA (omega -CTX), and N(G)-nitro-L-arginine methylester suppressed preferentially the mild hypoxic injury. The extracellular cyclic GMP formation, a marker of nitric oxide synthesis, was enhanced during hypoxia , although the extent was greater in the absence of glucose. As observed in the hypoxic injury, NS-7 preferentially inhibited the cyclic GMP formation induced by severe hypoxic insults, whereas MK-801 or omega -CTX reduced it under mild hypoxic condition. When 30 to 50 mM KCl was applied to normoxic slices, a concentration-dependent increase in the extracellular cyclic GMP formation was observed. NS-7 blocked the cyclic GMP formation induced by 50 mM KCl but not by 30 to 40 mM KCl, whereas omega -CTX suppressed only the 30 mM KCl-evoked response. In primary neuronal culture, NS-7 reversed KCl-induced increase in intracellular Ca(2+) in which the inhibition was marked when the KCl concentration was increased. These findings suggest that NS-7, unlike other neuroprotective compounds used in this study, is more effective in severe hypoxic injury. The highly voltage-dependent Ca(2+) channel blockade may contribute to the mode of neuroprotective action of NS-7. Record Date Created: 20000621

14/7/16 (Item 16 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10580919 20176956 PMID: 10714496
Design and biological evaluation of non-peptide analogues of omega - conotoxin MVIIA.
Menzler S; Bikker JA; Suman-Chauhan N; Horwell DC
Parke-Davis Neuroscience Research Centre, Cambridge, UK.
Bioorganic & medicinal chemistry letters (ENGLAND) Feb 21 2000, 10 (4) p345-7, ISSN 0960-894X Journal Code: C8B Languages: ENGLISH Document type: Journal Article Record type: Completed
Omega - conotoxin MVIIA, a highly potent antagonist of the N-type voltage sensitive calcium channel , has shown utility in several models of pain and ischemia . We report a series of three alkylphenyl ether based analogues which mimic three key amino acids of the toxin. Two of the compounds have been found to exhibit IC50 values of 2.7 and 3.3 microM at the human N-type voltage sensitive calcium channel . Record Date Created: 20000515

14/7/22 (Item 22 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10328680 99107720 PMID: 9889329
The role of voltage-gated Ca2+ channels in anoxic injury of spinal cord white matter.
Imaizumi T; Kocsis JD; Waxman SG
Department of Neurology, Yale University School of Medicine, New Haven, CT 06516, USA.
Brain research (NETHERLANDS) Jan 30 1999, 817 (1-2) p84-92, ISSN 0006-8993 Journal Code: B5L
Languages: ENGLISH Document type: Journal Article Record type: Completed
Dorsal column axons of the rat spinal cord are partially protected from anoxic injury following blockade of voltage-sensitive Na+ channels and the Na+/–Ca2+ exchanger. To examine the potential contribution of voltage-gated Ca2+ channels to anoxic injury of spinal cord axons, we studied axonal conduction in rat dorsal columns in vitro following a 60-min period of anoxia . Glass microelectrodes were used to record field potentials from the dorsal columns following distal local surface stimulation. Perfusion solutions containing blockers of voltage-gated Ca2+ channels were introduced 60 min prior to onset of anoxia and continued until 10 min after reoxygenation. Pharmacological blocking agents which are relatively selective for L- (verapamil, diltiazem, nifedipine) and N- (omega - conotoxin GVIA) type calcium channels were significantly protective against anoxia -induced loss of conduction, as was non-specific block using divalent cations. Other Ca2+ channel blockers (neomycin and omega - conotoxin MVIIC) that affect multiple Ca2+ channel types were also neuroprotective. Ni2+, which preferentially blocks R-type Ca2+ channels more than T-type channels , was also protective in a dose-dependent manner. These data suggest that the influx of Ca2+, through L-, N- and possibly R-type voltage-gated Ca2+ channels , participates in the pathophysiology of the Ca2+-mediated injury of spinal cord axons that is triggered by anoxia . Copyright 1999 Elsevier Science B.V. Record Date Created: 19990312

14/7/24 (Item 24 from file: 155 DIALOG(R)File 155:MEDLINE(R)
10308042 98340841 PMID: 9675077
Hypoxia evokes catecholamine secretion from rat pheochromocytoma PC-12 cells.
Taylor SC; Peers C
Institute for Cardiovascular Research, University of Leeds, United Kingdom.
Biochemical and biophysical research communications (UNITED STATES) Jul 9 1998, 248 (1) p13-7, ISSN 0006-291X Journal Code: 9Y8 Languages: ENGLISH Document type: Journal Article Record type: Completed
We have monitored exocytosis of catecholamines from individual PC-12 cells by amperometry using carbon fiber microelectrodes in order to investigate possible secretory responses to acute hypoxia . In normoxia, no secretion was detected from cells perfused with a solution containing 5 mM K+. However, when [K+] was raised (10-100 mM), exocytotic events were observed. Hypoxia (PO2 11 mmHg) stimulated secretion from PC-12 cells, and in hypoxic conditions exocytosis was greater at each [K+] studied as compared with normoxia. Hypoxia -evoked secretion was abolished in Ca2+ free solutions containing 1 mM EGTA and by the non-specific Ca2+ channel blocker, Cd2+ (200 microM). Secretion was also largely inhibited by omega - conotoxin GVIA (1 microM). Exocytosis was also observed in normoxia when cells were exposed to tetraethylammonium (1-10 mM), but not 4-aminopyridine (3 mM). Our findings indicate that hypoxia evokes exocytosis via depolarization arising from inhibition of a TEA-sensitive K+ conductance, leading to Ca2+ influx primarily via N-type Ca2+ channels . Record Date Created: 19980806

14/7/25 (Item 25 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10298442 98122085 PMID: 9460753
Alterations in K+ evoked profiles of neurotransmitter and neuromodulator amino acids after focal ischemia -reperfusion.
Lo EH; Pierce AR; Matsumoto K; Kano T; Evans CJ; Newcomb R
Department of Neurology, Harvard Medical School, Massachusetts General Hospital, Charlestown 02129, USA.
Neuroscience (UNITED STATES) Mar 1998, 83 (2) p449-58, ISSN 0306-4522 Journal Code: NZR Contract/Grant No.: R29NS32806, NS, NINDS Languages: ENGLISH Document type: Journal Article Record type: Completed
Secondary elevations in extracellular amino acids occur during reperfusion after transient cerebral ischemia . The delayed accumulation of excitatory amino acids may contribute to the progressive development of neuronal injury. In this study, we explored the mechanisms that may be involved in this phenomenon. Microdialysis samples from probes located in rabbit cortex were analysed with a chiral amino acid procedure. Concentrations of neurotransmitters (L-Glu, GABA), N-methyl-D-aspartate receptor modulators (D-Ser, Gly), an inhibitory neuromodulator (Tau), the lipid component phosphoethanolamine, and L-Gln, L-Ser and L-Ala were measured. Depolarization via perfusion with potassium was used to assess the status of release/reuptake systems at 2 and 4 h reperfusion after 2 h transient focal ischemia . Background experiments classified potassium evoked responses as calcium dependent or calcium-independent by inclusion of 30 microM omega -conopeptide MVIIC or by inclusion of 20 mM magnesium and ommision of calcium. During ischemia , large elevations of almost all amino acids occurred. During reperfusion, secondary elevations in transmitter amino acids (L-Glu, GABA) and N-methyl-D-aspartate receptor modulators (D-Ser, Gly) occurred. Tau remained slightly elevated whereas the lipid component phosphoethanolamine remained high and stable during reperfusion. Reperfusion significantly potentiated the potassium response for amino acids with calcium-dependent responses (L-Glu and GABA). In contrast, calcium-independent responses (Tau, phosphoethanolamine, L-Gln) were significantly attenuated. Intermediate behavior was observed with Gly, while no potassium responses were observed for D-Ser, L-Ser or L-Ala. These data demonstrate that perturbations in evoked amino acid profiles after ischemia -reperfusion are selective. Reduction of calcium-independent responses implicate a general decline in efficacy of transporter mechanisms that restore transmembrane gradients of ions and transmitters. Decreased efficacy of transporter systems may reduce transmitter reuptake

and account for the amplified release of L-Glu and GABA, thus contributing to progressive neural dysfunction after cerebral ischemia . Record Date Created: 19980312

14/7/26 (Item 26 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10297988 98115509 PMID: 9455974
SNX-111, a novel, presynaptic N-type calcium channel antagonist, is neuroprotective against focal cerebral ischemia in rabbits.
Perez-Pinzon MA; Yenari MA; Sun GH; Kunis DM; Steinberg GK
Department of Neurosurgery and Stanford Stroke Center, Stanford University Medical Center, CA 94305, USA.
Journal of the neurological sciences (NETHERLANDS) Dec 9 1997, 153 (1) p25-31, ISSN 0022-510X Journal Code: JBJ
Contract/Grant No.: K08 NS01860, NS, NINDS; RO1 NS 27292, NS, NINDS Languages: ENGLISH Document type: Journal Article Record type: Completed
Cytosolic Ca2+ overload has been proposed as a main cause of neuronal injury during cerebral ischemia . SNX-111, a synthetic product of the naturally occurring omega - conotoxin MVIIA, is a novel, presynaptic N-type Ca2+ channel antagonist and has been reported to be neuroprotective against cerebral ischemia . We studied the neuroprotective effects of SNX-111 in a rabbit model of focal cerebral ischemia . New Zealand white male rabbits (2.5-3.5 kg) were given 1 mg/kg/h i.v. SNX-111 (n=8) or normal saline (n=8) 10 min after onset of a 2-h period of transient focal cerebral ischemia induced by occlusion of the left middle cerebral, anterior cerebral and internal carotid arteries followed by 4 h reperfusion. SNX-111 significantly attenuated overall cortical ischemic neuronal damage by 44% (saline, 38.7+/-3.0%; SNX-111, 21.5+/-6.0%, P<0.05) and regions of hyperintensity on T2-weighted MRI by 30% (saline, 70.6+/-4.0%; SNX-111, 49.3+/-11.0%, P<0.05). No significant difference in (regional cerebral blood flow) rCBF or MAP (mean arterial blood pressure) was found between SNX-111- and saline-treated rabbits suggesting that neuroprotection is due to a cellular effect. We conclude that SNX-111 reduces ischemic injury in this model. Its use as a clinical neuroprotective agent for cerebrovascular surgery or stroke should be investigated further. Record Date Created: 19980305

14/7/28 (Item 28 from file: 155) DIALOG(R)File 155:MEDLINE(R)
09954079 98420452 PMID: 9750004
A novel Na+/Ca2+ channel blocker, NS-7, suppresses hypoxic injury in rat cerebrocortical slices.
Tatsumi S; Itoh Y; Ukai Y; Kimura K
Research Laboratories, Nippon Shinyaku Co., Ltd., Kyoto, Japan.
Naunyn-Schmiedeberg's archives of pharmacology (GERMANY) Aug 1998, 358 (2) p191-6, ISSN 0028-1298 Journal Code: NTQ Languages: ENGLISH Document type: Journal Article Record type: Completed
The substance 4-(4-fluorophenyl)-2-methyl-6-(5-piperidinopentyloxy) pyrimidine hydrochloride (NS-7) has been developed recently as a cerebroprotective compound with Na+ and Ca2+ channel blocking action. In the present study, the effect of NS-7 in an in vitro model of hypoxic injury was examined and the possible involvement of Na+ and Ca2+ channels in the hypoxic injury subsequently determined. When slices of rat cerebral cortex were exposed to hypoxia /glucose deprivation followed by reoxygenation and restoration of the glucose supply, marked leakage of lactate dehydrogenase (LDH) occurred 3-6 h after reoxygenation. This hypoxia /reoxygenation-induced injury was blocked almost completely by the removal of extracellular Ca2+ or by chelating intracellular Ca2+ with 1,2-bis(o-aminophenoxy)ethane-N,N,N',N'-tetraacetic acid tetra(acetoxymethy l)ester (BAPTA/AM). In addition, combined treatment with the N-type Ca2+ channel blocker omega - conotoxin GVIA and the P/Q-type Ca2+ channel blocker omega -agatoxin IVA significantly reduced LDH leakage, although neither of these Ca2+ channel blockers alone, nor nimodipine, an L-type Ca2+ channel blocker, was effective. On the other hand, several Na+ channel blockers, including tetrodotoxin, local anaesthetics and antiepileptics, significantly reduced the hypoxic injury. NS-7 (3-30 microM) concentration-dependently inhibited LDH leakage caused by hypoxia /reoxygenation, but had no influence on the reduction of tissue ATP content and energy charge during hypoxia and glucose deprivation. It is suggested that blockade of Na+ and Ca2+ channels is implicated in the cerebroprotective action of NS-7. Record Date Created: 19981204

14/7/32 (Item 32 from file: 155) DIALOG(R)File 155:MEDLINE(R)
09474308 97432733 PMID: 9286613
Effects of Ca2+ and Na+ channel inhibitors in vitro and in global cerebral ischaemia in vivo.
O'Neill MJ; Bath CP; Dell CP; Hicks CA; Gilmore J; Ambler SJ; Ward MA; Bleakman D
Eli Lilly and Company Ltd., Lilly Research Centre, Windlesham, Surrey, United Kingdom. oneill michael j@lilly com
European journal of pharmacology (NETHERLANDS) Aug 6 1997, 332 (2) p121-31, ISSN 0014-2999 Journal Code: EN6
Languages: ENGLISH Document type: Journal Article Record type: Completed
In the present study we have examined the effects of the small organic molecules: NNC 09-0026 ((-)-trans-1-butyl-4-(4-dimethylaminophenyl)-3-[(4-t rifluoromethyl-ph eno xy) methyl] piperidine dihydrochloride); SB 201823-A (4-[2-(3,4-dichlorophenoxy)ethyl]-1-pentyl piperidine hydrochloride); NS 649 (2-amino-1-(2,5-dimethoxyphenyl)-5-trifluoromethyl benzimidazole); CNS 1237 (N-acenaphthyl-N'-4-methoxynaphth-1-yl guanidine) and riluzole on human omega - conotoxin sensitive N-type voltage-dependent Ca2+ channel currents (ICa) expressed in HEK293 cells, on Na+ channel currents (INa) in acutely isolated cerebellar Purkinje neurones in vitro and in the gerbil model of global cerebral ischaemia in vivo. Estimated IC50 values for steady-state inhibition of ICa were as follows; NNC 09-0026, 1.1 microM; CNS 1237, 4.2 microM; SB 201823-A, 11.2 microM; NS 649, 45.7 microM and riluzole, 233 microM. Estimated IC50 values for steady-state inhibition of Na+ channel currents were as follows: NNC 09-0026, 9.8 microM; CNS 1237, 2.5 microM; SB 201823-A, 4.6 microM; NS 649, 36.7 microM and riluzole, 9.4 microM. In the gerbil model of global cerebral ischaemia the number of viable cells (mean +/- S.E.M.) per 1 mm of the CA1 was 215 +/- 7 (sham operated), 10 +/- 2 (ischaemic control), 44 +/- 15 (NNC 09-0026 30 mg/kg i.p.), 49 +/- 19 (CNS 1237 30 mg/kg i.p.), 11 +/- 2 (SB 201823-A 10 mg/kg i.p.), 17 +/- 4 (NS 649 50 mg/kg i.p.) and 48 +/- 18 (riluzole 10 mg/kg i.p.). Thus NNC 09-0026, CNS 1237 and riluzole provided significant neuroprotection when administered prior to occlusion while SB 201823-A and NS 649 failed to protect. These results indicate that the Ca2+ channel antagonists studied not only inhibited human N-type voltage-dependent Ca2+ channels but were also effective blockers of rat Na+ channels . Both NNC 09-0026 and CNS 1237 showed good activity at both Ca2+ and Na+ channels and this may contribute to the observed neuroprotection. Record Date Created: 19971215

14/7/33 (Item 33 from file: 155) DIALOG(R)File 155:MEDLINE(R)
09469299 97375400 PMID: 9231717
Selective changes in cell bodies and growth cones of nerve growth factor-differentiated PC12 cells induced by chemical hypoxia .
Gibson G; Toral-Barza L; Zhang H
Cornell University Medical College, Burke Medical Research Institute, White Plains, New York 10605, USA.
Journal of neurochemistry (UNITED STATES) Aug 1997, 69 (2) p603-11, ISSN 0022-3042 Journal Code: JAV Contract/Grant No.: AG11921, AG, NIA; AG14600, AG, NIA Languages: ENGLISH Document type: Journal Article Record type: Completed
Cytosolic free Ca2+ concentration ([Ca2+]i) was measured in differentiated PC12 cells to test whether chemical hypoxia selectively alters intracellular Ca2+ in growth cones and cell bodies. Hypoxia increased [Ca2+]i and exaggerated its response to K+ depolarization in both parts of the cells. [Ca2+]i in the cell bodies was greater than that in the growth cones under resting conditions and in response to K+ or hypoxia . Ca2+ channel blockers selectively altered these responses. The L- channel blocker nifedipine reduced [Ca2+]i following K+ depolarization by 67% in the cell bodies but only 25% in the growth cones. In contrast, the N- channel blocker omega - conotoxin GVIA (omega -CgTX) diminished K+-induced changes in [Ca2+]i only in the growth cones. During hypoxia , nifedipine was more effective in the cell bodies than in the growth cones. During hypoxia omega -CgTX diminished K+-induced changes by 50-75% in both parts of the cell, but only immediately after depolarization. The combination of nifedipine and omega -CgTX diminished the [Ca2+]i response to K+ with or without hypoxia by >90% in the cell body and 70% in the growth cones. Thus, the increased Ca2+ entry with K+ during hypoxia is primarily through L channels in the cell bodies, whereas in growth cones influx through L and N channels is about equal. The results show that chemical hypoxia selectively alters Ca2+ regulation in the growth cone and cell body of the same cell. Record Date Created: 19970812

14/7/38 (Item 38 from file: 155) DIALOG(R)File 155:MEDLINE(R)
09331614 97270322 PMID: 9125405
Identification of calcium channels involved in neuronal injury in rat hippocampal slices subjected to oxygen and glucose deprivation.
Small DL; Monette R; Buchan AM; Morley P
Institute for Biological Sciences, National Research Council of Canada, Ottawa. dan.small@nrc.ca
Brain research (NETHERLANDS) Apr 11 1997, 753 (2) p209-18, ISSN 0006-8993 Journal Code: B5L
Languages: ENGLISH Document type: Journal Article Record type: Completed
The presynaptic Ca2+-influx affecting glutamate release during neuropathological processes is mediated via voltage-sensitive calcium channels (VSCCs). There is controversy, however, over the fractional contribution of the specific channel types involved. We have addressed this by investigating the protective effects of various VSCC blockers on oxygen and glucose-deprived rat hippocampal slices. The viability of treated and non-treated slices was assayed electrophysiologically by measuring the evoked population spike (PS) amplitude in the stratum pyramidale of the CA1 region and by imaging slices loaded with fluorochrome dyes specific for dead (ethidium homodimer) and live (calcein) cells using confocal microscopy. PS amplitudes were significantly ($P < 0.01$) depressed from 4.4 +/- 0.2 mV (n = 38) to 0.2 +/- 0.1 mV (n = 40) after the deprivation insult. Responses from deprived slices treated with omega - conotoxin MVIIC (100 nM; 4.2 +/- 0.5 mV; n = 20) were not significantly different from control, non-deprived slice responses. In contrast, deprived slices treated with either L-type (0.1 or 1 microM nimodipine) or N-type (0.1 or 3 microM omega - conotoxin MVIIA) blockers showed no significant protection. The viability of CA1 neurons as revealed by the fluorescence live/dead confocal viability assay was consistent with the electrophysiological measurements. By comparison with previous studies using P- and Q-type blockers to attempt neuroprotection against the same deprivation insult, the rank order in which specific Ca2+- channel types contribute to neuronal death due to oxygen and glucose deprivation was determined to be Q > N >> P > L. Record Date Created: 19970623

14/7/40 (Item 40 from file: 155) DIALOG(R)File 155:MEDLINE(R)
09248788 97182239 PMID: 9030285
Ca2+ channel currents in type I carotid body cells from normoxic and chronically hypoxic rats.
Carpenter E; Wyatt CN; Hatton CJ; Bee D; Peers C
Institute for Cardiovascular Research, Leeds University, UK.
Advances in experimental medicine and biology (UNITED STATES) 1996, 410 p105-8, ISSN 0065-2598 Journal Code: 2LU Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19970605

14/7/45 (Item 45 from file: 155) DIALOG(R)File 155:MEDLINE(R)

09222432 96271052 PMID: 8926628
Characterization of a chemical anoxia model in cerebellar granule neurons using sodium azide: protection by nifedipine and MK-801.
Varming T; Drejer J; Frandsen A; Schousboe A
NeuroSearch A/S, Glostrup, Denmark.
Journal of neuroscience research (UNITED STATES) Apr 1 1996, 44 (1) p40-6, ISSN 0360-4012 Journal Code: KAC
Languages: ENGLISH Document type: Journal Article Record type: Completed
Induction of chemical anoxia , using sodium azide in cerebellar granule cells maintained in primary culture, was evaluated as an in vitro assay for screening of potential neuroprotective compounds. The purpose of this study was to evaluate sodium azide as an alternative to cyanide salts, compounds which, despite their unfavorable characteristics, are often used in assays for chemical anoxia . The viability of neuronal cultures after treatment with azide, with or without preincubation with calcium channel blockers, tetrodotoxin (TTX), or glutamate receptor antagonists, was monitored by subsequent incubation with the tetrazolium dye MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide), followed by isopropanol extraction and spectrophotometric quantification of cellularly reduced MTT. The azide-induced degeneration of neurons was shown to be dependent on the concentration as well as on the duration of incubation with submaximal concentrations of azide. Incubation of the neurons with nifedipine, a blocker of L-type voltage-sensitive calcium channels (L-VSCC), or with the noncompetitive N-methyl-D-aspartate (NMDA) subtype glutamate receptor antagonist MK-801, prior to addition of submaximal concentrations of azide, significantly attenuated azide-induced neuronal death. Blockers of N-type and Q-type VSCC (omega - conotoxin MVIIA and MVIIC, respectively) and the P-type VSCC blocker omega -agatoxin IVA had no effect in this assay. The sodium channel blocker TTX was without effect when added to neurons under depolarizing conditions, but potently and effectively protected cells when experiments were performed in a nondepolarizing buffer. The results show that chemical anoxia induced by incubation of cultured neurons with azide leads to detrimental effects, which may be quantitatively monitored by the capability of the cells to reduce MTT. This procedure is a suitable method for screening of compounds for possible protective effects against neuronal death induced by energy depletion. In addition, the results suggest involvement of L-type VSCC as well as of glutamate receptors in the pathways leading to neuronal degradation induced by energy depletion in cerebellar granule neurons. This would further support the notion that these pathways might be important in neurodegeneration induced by cerebral ischemia or anoxia . Record Date Created: 19961127

14/7/47 (Item 47 from file: 155) DIALOG(R)File 155:MEDLINE(R)
09056734 97054529 PMID: 8898826
Selective N-type calcium channel antagonist omega conotoxin MVIIA is neuroprotective against hypoxic neurodegeneration in organotypic hippocampal-slice cultures.
Pringle AK; Benham CD; Sim L; Kennedy J; Iannotti F; Sundstrom LE
Department of Clinical Neurological Sciences, University of Southampton, Southampton General Hospital, UK.
Stroke (UNITED STATES) Nov 1996, 27 (11) p2124-30, ISSN 0039-2499 Journal Code: V2J Languages: ENGLISH
Document type: Journal Article Record type: Completed
BACKGROUND AND PURPOSE: Neuroprotection by antagonists of both L-type and N-type calcium channels occurs in in vivo models of ischemia . The site of action of calcium channel antagonists is unclear, however, and it is likely that a combination of vascular and direct neuronal actions occurs. We have investigated the effects of blocking neuronal calcium channels using an organotypic hippocampal-slice model of ischemia . METHODS: Organotypic hippocampal-slice cultures prepared from 10-day-old rats were maintained in vitro for 14 days. Cultures were exposed to either 3 hours of oxygen deprivation (hypoxia) or 1 hour of combined oxygen and glucose deprivation (ischemia). Neuronal damage was quantified after 24 hours by propidium iodide fluorescence. RESULTS: Three hours of anoxia produced damage exclusively in CAT pyramidal cells. This damage was prevented by preincubation with omega conotoxin MVIIA, a selective N-type calcium channel blocker, and omega conotoxin MVIIC, which blocks N-type and other presynaptic neuronal calcium channels . The dihydropyridine nifedipine and the mixed calcium channel blocker SB201823-A were not protective. Furthermore, if addition of conotoxin MVIIA was delayed until after the hypoxic episode, a dose-dependent neuroprotective effect was observed, with an IC50 of 50 nmol/L. In contrast to hypoxia , none of the compounds was neuroprotective in the model of oxygen-glucose deprivation, although it was determined that extracellular calcium was essential for the generation of ischemic damage. CONCLUSIONS: These studies present clear evidence that neuroprotection by selective N-type calcium channel antagonists is mediated directly through neuronal calcium channels . In contrast, the neuroprotective effects of dihydropyridines may be mediated through vascular calcium channels or indirectly through actions in other brain regions. Record Date Created: 19961205

14/7/48 (Item 48 from file: 155) DIALOG(R)File 155:MEDLINE(R)
08907002 96106523 PMID: 8548294
Transient brain ischemia in rabbits: the effect of omega –conopeptide MVIIC on hippocampal excitatory amino acids.
Wu G; Kim HK; Zornow MH
Department of Anesthesiology, University of Texas Medical Branch, Galveston 77555-0830, USA.
Brain research (NETHERLANDS) Sep 18 1995, 692 (1-2) p118-22, ISSN 0006-8993 Journal Code: B5L Contract/Grant No.: RO1-NS29403, NS, NINDS Languages: ENGLISH Document type: Journal Article Record type: Completed
Neurologic injury that occurs after ischemia results from a cascade of events involving the release of various endogenous neurotoxins. A portion of the release of excitatory neurotransmitters is calcium dependent and may be attenuated by administration of calcium channel blockers. Using an in vivo model of ischemia , we studied the effects of omega -conopeptide MVIIC, a voltage-sensitive calcium channel blocker, and hypothermia (32 degrees C) on hippocampal glutamate and aspartate release in the peri-ischemic period. Thirty-four New Zealand white rabbits of either sex were anesthetized with halothane, intubated, and mechanically ventilated. Monitored variables included blood gases, mean arterial blood pressure, and the electroencephalogram. Microdialysis catheters were transversely inserted through the anterior portion of the dorsal hippocampus and perfused with artificial cerebrospinal fluid at a rate of 2 microliters/min. After stabilization period, animals were randomly assigned to one of the following groups: Control group (n = 8), 10 microM omega -conopeptide MVIIC group (n = 7), 100 microM omega -conopeptide MVIIC group (n = 7), Hypothermia group (n = 6; cranial temperature = 32 degrees C), and omega -conopeptide MVIIC + hypothermia group (n = 6; 100 microM omega -conopeptide MVIIC and cranial temperature 32 degrees C). All the rabbits were subjected to 10 minutes of global cerebral ischemia produced by neck tourniquet inflation combined with hypotension during halothane anesthesia. Conopeptide MVIIC was administered in the artificial cerebrospinal fluid used to perfuse the microdialysis catheter. In control animals, ischemia caused a significant increase in glutamate (9.7 fold) and aspartate (11.3 fold) concentrations.(ABSTRACT TRUNCATED AT 250 WORDS) Record Date Created: 19960220

14/7/49 (Item 49 from file: 155) DIALOG(R)File 155:MEDLINE(R)
08904087 96063291 PMID: 7472338
Voltage-gated calcium channels in CNS white matter: role in anoxic injury.
Fern R; Ransom BR; Waxman SG
Department of Neurology, Yale University School of Medicine, New Haven 06510, USA.
Journal of neurophysiology (UNITED STATES) Jul 1995, 74 (1) p369-77, ISSN 0022-3077 Journal Code: JC7
Languages: ENGLISH Document type: Journal Article Record type: Completed
1. The effect of Ca2+ channel antagonists on the extent of anoxia -induced white matter injury was studied in the rat optic nerve, a white matter tract. Compound action potentials (CAPs) were recorded before and after a standard 60-min anoxic period to assess the extent of anoxic injury. 2. The L-type Ca2+ channel antagonists verapamil (90 microM), diltiazem (50 microM), and nifedepine (2.5 microM) significantly protected the rat optic nerve from anoxic injury. Mean recovery of CAP area was 51.3 +/- 3.0% (mean +/- SE, n = 8, P < 0.01), 65.6 +/- 5.3% (n = 8, P < 0.01), and 54.3 +/- 6.1% (n = 8, P < 0.01), respectively. Mean CAP recovery under control conditions was 35.2 +/- 0.3 (n = 33). 3. Simultaneous block of L-type and N-type Ca2+ channels by coapplication of 50 microM diltiazem and 1 microM SNX-124 [synthetic omega - conotoxin (CgTx) GVIA], resulted in postanoxic CAP recovery of 73.6 +/- 6.0% (n = 12), significantly larger than CAP recovery in diltiazem alone (P < 0.001). Block of CgTx MVIIC-sensitive channels in addition to L-type and N-type channels by coapplication of 50 microM diltiazem + 1 microM SNX-230 + 1 microM SNX-124 failed to produce any additional increase in CAP recovery (71.3 +/- 5.6%, n = 8). Application of 1 microM SNX-124 alone did not significantly protect against anoxic injury (CAP recovery, 36.3 +/- 2.9%, n = 10).(ABSTRACT TRUNCATED AT 250 WORDS) Record Date Created: 19951129

14/7/52 (Item 52 from file: 155) DIALOG(R)File 155:MEDLINE(R)
08858461 94309792 PMID: 8035911
Omega - conotoxin GVIA protects against ischemia -induced neuronal death in the Mongolian gerbil but not against quinolinic acid-induced neurotoxicity in the rat.
Yamada K; Teraoka T; Morita S; Hasegawa T; Nabeshima T
Department of Neuropsychopharmacology, Nagoya University School of Medicine, Japan.
Neuropharmacology (ENGLAND) Feb 1994, 33 (2) p251-4, ISSN 0028-3908 Journal Code: NZB
Languages: ENGLISH Document type: Journal Article Record type: Completed
Excessive release of neurotransmitters is reported to contribute to the delayed neuronal death in animal models of cerebral ischemia . Since evidence is accumulating that N-type voltage-sensitive calcium channels (N- channels) regulate the release of neurotransmitters, we investigated the effects of omega - conotoxin GVIA (omega -CTX), an antagonist of N- channels , on the delayed neuronal death following transient ischemia in gerbils. Delayed neuronal death in the CA1 subfield of the hippocampus following 5-min ischemia was attenuated by omega -CTX in a dose-dependent manner when the agent was injected intracisternally 1 hr before ischemia was produced. However, omega -CTX failed to prevent neurotoxicity produced by a direct injection of quinolinic acid into the hippocampus in rats. These results suggest that omega -CTX has a neuroprotective effect against ischemic brain injury, which effect probably results from its inhibition of the excessive release of neurotransmitters, including excitatory amino acids, during ischemia . Record Date Created: 19940812

14/7/53 (Item 53 from file: 155) DIALOG(R)File 155:MEDLINE(R)
08858076 94302114 PMID: 8029306
Pharmacological profile of a novel neuronal calcium channel blocker includes reduced cerebral damage and neurological deficits in rat focal ischemia .
Barone FC; Price WJ; Jakobsen P; Sheardown MJ; Feuerstein G
Department of Cardiovascular Pharmacology, SmithKline Beecham Pharmaceuticals, King of Prussia, PA 19406.
Pharmacology, biochemistry, and behavior (UNITED STATES) May 1994, 48 (1) p77-85, ISSN 0091-3057 Journal Code: P3Q Languages: ENGLISH Document type: Journal Article Record type: Completed
Excessive calcium entry into depolarized neurons contributes significantly to cerebral tissue damage following ischemia . Therefore, blocking voltage-operated calcium channels on nerve cells should provide significant neuroprotection in ischemia .

We now report on a novel neuronal calcium channel blocker, NNC 09-0026, in terms of its selective effects on neuronal calcium current and its efficacy in reducing infarct size and neurological deficits in a rat model of focal stroke. In the present studies, the effects of NNC 09-0026 on neuronal calcium influx, calcium channel binding, and cardiovascular parameters were determined. Also, phencyclidine, NNC 09-0026, or vehicle were administered i.v. to rats subjected to permanent middle cerebral and common carotid artery occlusions. Infarct volumes and contralateral forepaw and hindlimb neurological deficits were assessed at 24 and 48 h after onset of stroke. NNC 09-0026 exhibited a pharmacological profile suggesting selectivity at neuronal calcium channels . It inhibited potassium-stimulated calcium uptake into rat synaptosomes with an IC50 of 13 microM. Voltage-operated calcium currents measured from cultured rat dorsal root ganglion cells using the patch clamp technique were blocked by 43% at 10 microM ($p < 0.05$). The compound showed only weak effects on smooth muscle from the guinea pig taenia coli and was relatively inactive at displacing nitrendipine and omega - conotoxin in receptor-binding studies. Single, bolus injections of NNC 09-0026 as high as 10 mg/kg i.v. produced only 12% reduction in heart rate and a 28% decrease in blood pressure.(ABSTRACT TRUNCATED AT 250 WORDS) Record Date Created: 19940810

14/7/54 (Item 54 from file: 155) DIALOG(R)File 155:MEDLINE(R)
08651525 96062914 PMID: 7473774
Two different mechanisms of noradrenaline release during normoxia and simulated ischemia in human cardiac tissue.
Kurz T; Richardt G; Hagl S; Seyfarth M; Schomig A
Department of Medicine I, Technical University, Munich, Germany.
Journal of molecular and cellular cardiology (ENGLAND) May 1995, 27 (5) p1161-72, ISSN 0022-2828 Journal Code: J72 Languages: ENGLISH Document type: Journal Article Record type: Completed
Species-related differences in the mechanisms of noradrenaline release during normoxia and myocardial ischemia emphasize the need for studies on human hearts. Therefore, the mechanisms of noradrenaline release were investigated during normoxia and energy depletion in incubated human atrial tissue and compared to the release characteristics in normoxic and ischemic rat heart. Potential differences of atrial versus ventricular myocardium were assessed by comparing catecholamine release during electrical stimulation and ischemia in isolated rat atrium with release characteristics in the intact perfused heart. The overflow of endogenous noradrenaline and its deaminated metabolite dihydroxyphenylethyleneglycol (DOPEG) were determined by high pressure liquid chromatography and electrochemical detection. During normoxia noradrenaline release was evoked by electrical field stimulation. Stimulation-induced noradrenaline release depended on the extracellular calcium concentration in both species and was almost completely suppressed under calcium-free conditions. The release was significantly inhibited by neuronal (N-type) calcium channel blockers such as omega - conotoxin (100 nmol/l) and cadmium chloride (100 mumol/l), indicating a predominant role of N-type calcium channels in exocytotic noradrenaline release from sympathetic neurons in human and rat heart. Desipramine (100 nmol/l) enhanced the overflow of noradrenaline evoked by electrical stimulation in both species by blocking neuronal catecholamine uptake (uptake1). Myocardial ischemia was caused by interruption of perfusion flow in rat heart and simulated by anoxic and glucose-free incubation in human and rat atrial tissue. Ischemia - and anoxia -induced noradrenaline release in rat heart and human atrial tissue was unaffected by varying extracellular calcium concentrations and occurred even after omission of calcium and addition of EGTA (1 mmol/l). In both species neither omega - conotoxin (100 nmol/l) nor cadmium chloride (100 mumol/l) affected ischemia -induced noradrenaline overflow in both rat heart and atrium as well as in human atrium. In human and rat atrial tissue, blockade of energy metabolism in the presence of oxygen (cyanide model) resulted in a desipramine-sensitive release of noradrenaline, which was accompanied by DOPEG overflow, indicating increased axoplasmic noradrenaline concentration. The data imply a dual mechanism of noradrenaline release in the human heart. During normoxia noradrenaline release is modulated by neuronal calcium influx indicating exocytotic release. Ischemia -induced noradrenaline release, however, is independent of calcium and inhibited by uptake1 blockade suggesting nonexocytotic release mechanism. The characteristics of noradrenaline release in human atrial tissue provide evidence for carrier-mediated release of noradrenaline from sympathetic neurons operative in the ischemic human myocardium.

17/6/1 (Item 1 from file: 155) 06936924 94034338 PMID: 1364103
Ciguatoxic fish in the French West Indies. 1992

17/6/2 (Item 2 from file: 155) 06486062 88311800 PMID: 3409658
Bone lipids of Jamaican reef fishes. 1988

17/6/3 (Item 3 from file: 155) 05456541 91018806 PMID: 2485547
[Risk factors of ciguatera in the French West Indies in Saint-Barthelemy, Saint-Martin and Anguilla] Facteurs de risque ciguaterique aux Antilles dans la region de Saint-Barthelemy, Saint-Martin et Anguilla. 1989

17/6/4 (Item 4 from file: 155) 05034631 86272379 PMID: 3731371
[Evaluation of different-stage levels of ciguatera toxicity of the marine food fish chain found around Saint Barthelemy Island in French Antilles] Evaluation des niveaux de toxicite ciguaterique des differents etages de la chaine trophique pisciaire marine presente autour de l'ile de Saint-Barthelemy aux Antilles Francaises. 1986

17/6/5 (Item 1 from file: 5) 12968619 BIOSIS NO.: 200100175768
Growth, mortality and exploitation rate of Priacanthus arenatus (Perciformes: Priacanthidae), in the trawl fisheries of northeast Venezuela. ORIGINAL LANGUAGE TITLE: Crecimiento, mortalidad y tasa de explotacion de Priacanthus arenatus (Perciformes: Priacanthidae), en la pesca de arrastre del nororiente de Venezuela. 2000

17/6/6 (Item 2 from file: 5) 12938841 BIOSIS NO.: 200100145990
Mechanisms for evolving hypervariability: The case of conopeptides. 2001

17/6/7 (Item 3 from file: 5) 12825730 BIOSIS NO.: 200100032879
Helminths from Priacanthus arenatus Cuvier, 1829 (Pisces, Priacanthidae) in Cabo Frio, RJ, Brazil. ORIGINAL LANGUAGE TITLE: Helmintos parasitos de Priacanthus arenatus Cuvier, 1829 (Pisces, Priacanthidae) em Cabo Frio, RJ, Brasil. 2000

17/6/8 (Item 4 from file: 5) 11341206 BIOSIS NO.: 199800122538
New myxosporidian species of the genus Henne-guya Thelohan, 1895 (Myxozoa, Myxosporea) parasites of marine fishes from Senegal. Light and electron microscopic studies. 1997

17/6/9 (Item 5 from file: 5) 11024278 BIOSIS NO.: 199799645423
New species of Agrilus from Africa (Coleoptera Buprestidae).

17/6/10 (Item 6 from file: 5) 09994208 BIOSIS NO.: 199598449126
Distribution, abundance, and reproduction of Priacanthus arenatus Cuvier (Pisces: Priacanthidae) on the continental shelf in the southern Gulf of Mexico. 1995

17/6/11 (Item 7 from file: 5) 09407875 BIOSIS NO.: 199497416245
Demersal bony fish of the outer shelf and upper slope of the southern Brazil subtropical convergence ecosystem. 1994

17/6/12 (Item 8 from file: 5) 08236990 BIOSIS NO.: 000043025663
PRIACANTHIDAE 1990

17/6/13 (Item 9 from file: 5) 06862110 BIOSIS NO.: 000089011700
SKELETAL FATTY ACIDS IN FISH FROM DIFFERENT DEPTHS OFF JAMAICA WEST INDIES 1989

17/6/14 (Item 10 from file: 5) 06651681 BIOSIS NO.: 000087093858
REDESCRIPTION OF ONCOPHORA-MELANOCEPHALA RUDOLPHI 1819 BAUDIN-LAURENCIN 1971 NEMATODA CAMALLANIDAE 1988

17/6/15 (Item 11 from file: 5) 06582457 BIOSIS NO.: 000087024618
REVISION PHYLOGENY AND BIOGEOGRAPHIC COMMENTS ON THE CIRCUMTROPICAL MARINE PERCOID FISH FAMILY PRIACANTHIDAE 1988

17/6/16 (Item 12 from file: 5) 06258675 BIOSIS NO.: 000086092858
REDESCRIPTION OF ONCOPHORA-MELANOCEPHALA RUDOLPHI 1819 BAUDIN-LAURENCIN 1971 NEMATODA CAMALLANIDAE 1988

17/6/17 (Item 13 from file: 5) 06232250 BIOSIS NO.: 000086066432
BONE LIPIDS OF JAMAICAN REEF FISHES 1988

17/6/18 (Item 14 from file: 5) 04969553 BIOSIS NO.: 000031044685
ECHINODERMS OF THE CANTABRICO 83 EXPEDITION OFF ASTURIAS NORTH SPAIN 1985

17/6/19 (Item 15 from file: 5) 04870123 BIOSIS NO.: 000030063247
ON THE GENUS PODOSPHAERASTER ECHINODERMATA ASTEROIDEA WITH DESCRIPTION OF A NEW SPECIES FROM THE NORTH ATLANTIC 1985

17/6/20 (Item 16 from file: 5) 04737833 BIOSIS NO.: 000080040960
TRIASSIC FORAMINIFERA FROM SOUTHLAND SYNCLINE NEW-ZEALAND 1984

17/6/21 (Item 17 from file: 5) 04643573 BIOSIS NO.: 000079056610
REVISION OF EASTERN PACIFIC CATALUFAS PISCES PRIACANTHIDAE WITH DESCRIPTION OF A NEW GENUS AND DISCUSSION OF THE FOSSIL RECORD 1984

17/6/22 (Item 18 from file: 5) 04643572 BIOSIS NO.: 000079056609
MARINE AND FRESHWATER STINGRAYS DASYATIDAE OF WEST AFRICA WITH DESCRIPTION OF A NEW SPECIES 1984

17/6/23 (Item 19 from file: 5) 03653620 BIOSIS NO.: 000074069197
THE CADDIS-FLY GENUS SETODES IN NORTH AMERICA TRICHOPTERA LEPTOCERIDAE 1982

17/6/24 (Item 20 from file: 5) 03607574 BIOSIS NO.: 000074023151
2 NEW SPECIES AND 6 NEW RECORDS OF LABRID FISHES FROM THE RED SEA 1981

17/6/25 (Item 21 from file: 5) 03247233 BIOSIS NO.: 000071060344
ASTEROIDEA ECHINODERMATA FROM THE GUYANA SHELF 1979

17/6/26 (Item 22 from file: 5) 02626079 BIOSIS NO.: 000037014139
APPROXIMATE COMPOSITION OF SOME FISHES OF VENEZUELA 1976

17/6/27 (Item 23 from file: 5) 02621664 BIOSIS NO.: 000037009722
SOME DIGENETIC TREMATODES OF MARINE FISHES FROM THE BARRIER REEF AND REEF LAGOON OF BELIZE 1977

17/6/28 (Item 24 from file: 5) 02305445 BIOSIS NO.: 000015018960
NOTES ON TROPICAL MARINE FISHES IN ALABAMA WATERS WITH NEW RECORDS FOR THE REGION 1978

17/6/29 (Item 25 from file: 5) 01905118 BIOSIS NO.: 000061065212
FISHES OF THE PLIOCENE GLENNS FERRY FORMATION SOUTHWEST IDAHO USA 1975

17/6/30 (Item 26 from file: 5) 01548629 BIOSIS NO.: 000011048618
CONIDAE WITH SMOOTH AND GRANULATED SHELLS 1973

17/6/31 (Item 27 from file: 5) 01442852 BIOSIS NO.: 000058012822
HIRUNDICHTHYS-RONDELETI NEW-RECORD COOKEOLUS-BOOPS NEW-RECORD PRIACANTHUS- ARENATUS NEW-RECORD SERIOLA-DUMERILI NEW-RECORD 4 SPECIES NEW TO THE CANADIAN ATLANTIC ICHTHYO FAUNA 1973

17/6/32 (Item 28 from file: 5) 01394665 BIOSIS NO.: 000057034633
THE TAPETUM LUCIDUM IN THE EYE OF THE BIG-EYE PRIACANTHUS- ARENATUS 1973

17/6/33 (Item 29 from file: 5) 01186948 BIOSIS NO.: 000055067174
ORDER NEUROPTERA THE ENTOMO FAUNA OF THE CARAORMAN GRIND DANUBE DELTA 1972

17/6/34 (Item 30 from file: 5) 00914187 BIOSIS NO.: 000053034357
COMPARATIVE STUDY OF THE GILLS OF SOME MARINE PERCIFORMES 1970

17/6/35 (Item 31 from file: 5) 00733383 BIOSIS NO.: 000052093452
THE BRAIN CASE OF THE HOLOSTEAN FISH MACREPISTIUS WITH COMMENTS ON NEURO CRANIAL OSSIFICATION IN THE ACTINOPTERYGII 1971

17/6/36 (Item 32 from file: 5) 00589121 BIOSIS NO.: 000007039086
AN UNUSUALLY LARGE AGGREGATION OF PREJUVENILE BIGEYES PRIACANTHUS- ARENATUS IN THE WEST-INDIES 1971

17/6/37 (Item 33 from file: 5) 00143466 BIOSIS NO.: 000005043466
OCCURRENCE OF THE BIGEYE IN LONG-ISLAND NEW-YORK USA WATERS PRIACANTHUS- ARENATUS 1968

19/6/1 (Item 1 from file: 5) 03046214 BIOSIS NO.: 000070071832
COMPARATIVE MORPHOLOGY OF RADULAR TEETH IN CONUS OBSERVATIONS WITH SCANNING ELECTRON MICROSCOPY 1980

22/6/1 (Item 1 from file: 5) 12838771 BIOSIS NO.: 200100045920
Electrophysiological characteristics of the Ca2+-activated Cl- channel family of anion transport proteins. 2000

22/6/2 (Item 2 from file: 5) 09649234 BIOSIS NO.: 199598104152
A study of some unusual, well preserved Oligocene diatoms from Antarctica. BOOK TITLE: Supplement to Nova Hedwigia; Progress in diatom studies: Contributions to taxonomy, ecology and nomemclature ORIGINAL LANGUAGE BOOK TITLE: Beihefte zur Nova Hedwigia; Progress in diatom studies: Contributions to taxonomy, ecology and nomemclature. 1993

25/6/1 (Item 1 from file: 5) 02196651 BIOSIS NO.: 000064039170
PANDELETEIUS OF VENEZUELA AND COLOMBIA CURCULIONIDAE BRACHYDERINAE TANYMECINI 1976

25/6/2 (Item 2 from file: 5) 01971020 BIOSIS NO.: 000062061135
NEW SCOLYTIDAE AND PLATYPODIDAE FROM PAPUA AND NEW-GUINEA PART 4 NO 317 CONTRIBUTION TO THE MORPHOLOGY AND TAXONOMY OF THE SCOLYTOIDEA 1975

28/6/1 (Item 1 from file: 155) 12635892 21581542 PMID: 11724570
A new omega- conotoxin that targets N-type voltage-sensitive calcium channels with unusual specificity. Dec 4 2001

28/6/2 (Item 2 from file: 155) 10803520 99255390 PMID: 10320362
Biochemical characterization and nuclear magnetic resonance structure of novel alpha-conotoxins isolated from the venom of Conus consors . May 11 1999

28/6/3 (Item 3 from file: 155) 10579080 20258204 PMID: 10797869
A review on conotoxins targetin g ion channels and acetylcholine receptorsof the vertebrate neuromuscular junction. 1999

28/6/4 (Item 4 from file: 155) 10352107 99440133 PMID: 10510177
A new conotoxin isolated from Conus consors venom acting selectively on axons and motor nerve terminals through a Na+-dependent mechanism. Sep 1999

28/6/5 (Item 1 from file: 5) 13357793 BIOSIS NO.: 200100564942
7th Meetings in Toxinology: French Society for the Study of Toxins, Paris, France, December 2-3, 1999. 2000

28/6/6 (Item 2 from file: 5) 12630955 BIOSIS NO.: 200000384457
New conotoxins purified from piscivorous cone snails that discriminate between nerve and muscle voltage-gated sodium channels at the frog neuromuscular junction. 2000

28/7/1 (Item 1 from file: 155) DIALOG(R)File 155:MEDLINE(R)
12635892 21581542 PMID: 11724570
A new omega- conotoxin that targets N-type voltage-sensitive calcium channels with unusual specificity.
Favreau P; Gilles N; Lamthanh H; Bournaud R; Shimahara T; Bouet F; Laboute P; Letourneux Y; Menez A; Molgo J; Le Gall F
Institut Federatif de Neurobiologie Alfred Fessard, Laboratoire de Neurobiologie Cellulaire et Moleculaire, UPR 9040, CNRS, 91198 Gif sur Yvette Cedex, France.
Biochemistry (United States) Dec 4 2001, 40 (48) p14567-75, ISSN 0006-2960 Journal Code: 0370623 Languages: ENGLISH Document type: Journal Article Record type: In Process
A new specific voltage-sensitive calcium channel (VSCC) blocker has been isolated from the venom of the fish-hunting cone snail Conus consors . This peptide, named omega-Ctx CNVIIA, consists of 27 amino acid residues folded by 3 disulfide bridges. Interestingly, loop 4, which is supposed to be crucial for selectivity, shows an unusual sequence (SSSKGR). The synthesis of the linear peptide was performed using the Fmoc strategy, and the correct folding was achieved in the presence of guanidinium chloride, potassium buffer, and reduced/oxidized glutathione at 4 degrees C for 3 days. Both synthetic and native toxin caused an intense shaking activity, characteristic of omega-conotoxins targeting N-type VSCC when injected intracerebroventricularly to mice. Binding studies on rat brain synaptosomes revealed that the radioiodinated omega-Ctx CNVIIA specifically and reversibly binds to high-affinity sites with a K(d) of 36.3 pM. Its binding is competitive with omega-Ctx MVIIA at low concentration (K(i) = 2 pM). Moreover, omega-Ctx CNVIIA exhibits a clear selectivity for N-type VSCCs versus P/Q-type VSCCs targeted respectively by radioiodinated omega-Ctx GVIA and omega-Ctx MVIIC. Although omega-Ctx CNVIIA clearly blocked N-type Ca(2+) current in chromaffin cells, this toxin did not inhibit acetylcholine release evoked by nerve stimuli at the frog neuromuscular junction, in marked contrast to omega-Ctx GVIA. omega-Ctx CNVIIA thus represents a new selective tool for blocking N-type VSCC that displays a unique pharmacological profile and highlights the diversity of voltage-sensitive Ca(2+) channels in the animal kingdom. Record Date Created: 20011128

28/7/5 (Item 1 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2002 BIOSIS. All rts. reserv.
13357793 BIOSIS NO.: 200100564942
7th Meetings in Toxinology: French Society for the Study of Toxins, Paris, France, December 2-3, 1999.
AUTHOR: French Society for the Study of Toxins
JOURNAL: Toxicon 38 (12):p1629-1652 December, 2000 MEDIUM: print CONFERENCE/MEETING: 7th Meetings in Toxinology: French Society for the Study of Toxins Paris, France December 02-03, 1999 SPONSOR: French Society for the Study of Toxins ISSN: 0041-0101 RECORD TYPE: Abstract LANGUAGE: English; French SUMMARY LANGUAGE: English
ABSTRACT: This meeting contains abstracts of 34 papers, written in English and French, covering topics in toxin research (toxicology), including lymphocyte activation, superantigens, venoms, food toxins, neurotoxins, chemical defense, biotechnology, histochemistry, gene expression, NMR structural characterization, in vivo immunodetection, for animal and bacterial source toxins.

28/7/6 (Item 2 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2002 BIOSIS. All rts. reserv.
12630955 BIOSIS NO.: 200000384457
New conotoxins purified from piscivorous cone snails that discriminate between nerve and muscle voltage-gated sodium channels at the frog neuromuscular junction.
AUTHOR: Le Gall Frederic(a); Favreau Philippe(a); Benoit Evelyne(a); Mattei Cesar(a); Thanh Hung Lam; Bouet Francoise; Letourneux Yves(a); Menez Andre; Molgo Jordi(a)
AUTHOR ADDRESS: (a)Laboratoire de Neurobiologie Cellulaire et Moleculaire, U.P.R. 9040, CNRS, 91198, Gif-sur-Yvette Cedex**France
JOURNAL: Journal of Physiology (Cambridge) 525Pp78P June, 2000 MEDIUM: print CONFERENCE/MEETING: Meeting the Physiological Society London, England April 12-14, 2000 SPONSOR: The Physiological Society ISSN: 0022-3751
RECORD TYPE: Citation LANGUAGE: English SUMMARY LANGUAGE: English

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13357793 BIOSIS NO.: 200100564942
7th Meetings in Toxinology: French Society for the Study of Toxins, Paris, France, December 2-3, 1999.
AUTHOR: French Society for the Study of Toxins
JOURNAL: Toxicon 38 (12):p1629-1652 December, 2000 MEDIUM: print CONFERENCE/MEETING: 7th Meetings in Toxinology: French Society for the Study of Toxins Paris, France December 02-03, 1999 SPONSOR: French Society for the Study of Toxins ISSN: 0041-0101 RECORD TYPE: Abstract LANGUAGE: English; French SUMMARY LANGUAGE: English
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28/7/6 (Item 2 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2002 BIOSIS. All rts. reserv.
12630955 BIOSIS NO.: 200000384457

New conotoxins purified from piscivorous cone snails that discriminate between nerve and muscle voltage-gated sodium channels at the frog neuromuscular junction.
AUTHOR: Le Gall Frederic(a); Favreau Philippe(a); Benoit Evelyne(a); Mattei Cesar(a); Thanh Hung Lam; Bouet Francoise; Letourneux Yves(a); Menez Andre; Molgo Jordi(a)
AUTHOR ADDRESS: (a)Laboratoire de Neurobiologie Cellulaire et Moleculaire, U.P.R. 9040, CNRS, 91198, Gif-sur-Yvette Cedex**France
JOURNAL: Journal of Physiology (Cambridge) 525Pp78P June, 2000 MEDIUM: print CONFERENCE/MEETING: Meeting of the Physiological Society London, England April 12-14, 2000 SPONSOR: The Physiological Society ISSN: 0022-3751 RECORD TYPE: Citation LANGUAGE: English SUMMARY LANGUAGE: English

33/6/1 (Item 1 from file: 155) 09570493 97426393 PMID: 9278406
Differential targeting of nicotinic acetylcholine receptors by novel alphaA-conotoxins. Sep 5 1997

33/6/2 (Item 2 from file: 155) 08637641 96062516 PMID: 7578057
alpha- Conotoxin EI, a new nicotinic acetylcholine receptor antagonist with novel selectivity. Nov 7 1995

33/6/3 (Item 1 from file: 5) 13520749 BIOSIS NO.: 200200149570
Solution conformation of alpha- conotoxin EI, a neuromuscular toxin specific for the alpha1/delta subunit interface of Torpedo nicotinic acetylcholine receptor. 2001

33/6/4 (Item 2 from file: 5) 12630955 BIOSIS NO.: 200000384457
New conotoxins purified from piscivorous cone snails that discriminate between nerve and muscle voltage-gated sodium channels at the frog neuromuscular junction. 2000

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Three-dimensional structure of alpha- conotoxin EI determined by 1H NMR spectroscopy. 1999

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Alpha- Conotoxin E-I, a new nicotinic acetylcholine receptor antagonist with novel selectivity. 1995

37/6/1 (Item 1 from file: 155) 11740706 21488774 PMID: 11602328
Histological demonstration of voltage dependent calcium channels on calcitonin gene-related peptide-immunoreactive nerve fibres in the mouse knee joint. Oct 26 2001

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Differential involvement of conotoxin -sensitive mechanisms in neurogenic vasodilatation responses: effects of age. Aug 2001

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Refined solution structure of omega - conotoxin GVIA: implications for calcium channel binding. Mar 1999

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Role of disulfide bridges in the folding, structure and biological activity of omega - conotoxin GVIA. Sep 14 1999

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Conotoxin TVIIA, a novel peptide from the venom of Conus tulipa 2. Three-dimensional solution structure. Aug 2000

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Conotoxin TVIIA, a novel p eptide from the venom of Conus tulipa 1. Isolation, characterization and chemical synthesis. Aug 2000

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Cannabinoid CB1 receptor-mediated inhibition of prolactin release and signaling mechanisms in GH4C1 cells. May 2000

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A molecular mechanism for toxin block in N-type calcium channels. Feb 1998

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Synthesis and biological characterization of a series of analogues of omega - conotoxin GVIA. Nov-Dec 1995

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A common structural motif incorporating a cystine knot and a triple-stranded beta-sheet in toxic and inhibitory polypeptides. Oct 1994

37/6/11 (Item 11 from file: 155) 08857093 94279504 PMID: 8010158
Different omega -conotoxins mark the development of Swiss Webster mouse cortex suggesting N-type voltage sensitive calcium channel subtypes. Feb 1994

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Characterization of the binding of omega -conopeptides to different classes of non-L-type neuronal calcium channels. Jun 1994

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Three-dimensional structure in solution of the calcium channel blocker omega - conotoxin . Nov 20 1993

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Evidence for sympathetic neurotransmission through presynaptic N-type calcium channels in human saphenous vein. Sep 1993

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Omega - conotoxin -sensitive calcium channels modulate autonomic neurotransmission in guinea pig airways. Jan 1992

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Molecular cloning of the alpha-1 subunit of an omega - conotoxin -sensitive calcium channel. Jun 1 1992

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Precursor structure of omega - conotoxin GVIA determined from a cDNA clone. Sep 1992

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A new Conus peptide ligand for mammalian presynaptic Ca2+ channels. Jul 1992

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Monoclonal antibodies against the presynaptic calcium channel antagonist omega - conotoxin GVI A from cone snail poison. Feb 12 1990

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Calcium channels in solitary retinal ganglion cells from post-natal rat. Nov 1989

37/6/21 (Item 21 from file: 155) 06057757 89314280 PMID: 2546090
Omega - conotoxin GVIA specifically blocks neuronal mechanisms in rat ileum. May-Jun 1989

37/6/22 (Item 22 from file: 155) 06028844 88318603 PMID: 2457794
Characterization of the omega - conotoxin -binding molecule in rat brain synaptosomes and cultured neurons. Aug 1988

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Effects of synthetic omega - conotoxin GVIA (omega -CgTX GVIA) on the membrane calcium current of an identifiable giant neurone, d-RPLN, of an African giant snail (Achatina fulica Ferussac), measured under the voltage clamp condition. 1987

37/6/24 (Item 24 from file: 155) 06018852 87231999 PMID: 2438698
Omega - conotoxin : direct and persistent blockade of specific types of calcium channels in neurons but not muscle. Jun 1987

37/6/25 (Item 25 from file: 155) 06014177 87299637 PMID: 2441741
Neuronal calcium channel antagonists. Discrimination between calcium channel subtypes using omega - conotoxin from Conus magus venom. Apr 21 1987

37/6/26 (Item 26 from file: 155) 06013786 87282599 PMID: 3112325
Transmitter release from presynaptic terminals of electric organ: inhibition by the calcium channel antagonist omega Conus toxin. Aug 1987

37/6/27 (Item 27 from file: 155) 06012209 87218818 PMID: 3034633
Effects of synthetic omega - conotoxin on synaptic transmission. Mar 31 1987

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Omega Conus geographus toxin: a peptide that blocks calcium channels. Apr 20 1987

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Characterization of the omega - conotoxin target. Evidence for tissue-specific heterogeneity in calcium channel types. Feb 10 1987

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Neuronal calcium channel inhibitors. Synthesis of omega - conotoxin GVIA and effects on 45Ca uptake by synaptosomes. Jan 25 1987

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Intrasynaptosomal free calcium concentration is increased by phorbol esters via a 1,4-dihydropyridine-sensitive (L-type) Ca2+ channel. Mar 14 1989

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Purification and sequence of a presynaptic peptide toxin from Conus geographus venom. Oct 23 1984

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A venom peptide with a novel presynaptic blocking action. Mar 15-21 1984

37/6/34 (Item 1 from file: 5) 10340307 BIOSIS NO.: 199698795225
Conotoxin -sensitive and conotoxin -resistant Ca-2+ currents in fish retinal ganglion cells. 1996

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Omega - conotoxin GVIA blocks nicotine-induced catecholamine secretion by blocking the nicotinic receptor-activated inward currents in bovine chromaffin cells. 1995

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Inhibition by omega - conotoxin GVIA of adrenal catecholamine release in response to endogenous and exogenous acetylcholine. 1994

37/6/37 (Item 4 from file: 5) 09004089 BIOSIS NO.: 199497012459
Characteristics of (125I) omega - conotoxin MVIIA binding to rat neocortical membranes. 1993

37/6/38 (Item 5 from file: 5) 08860105 BIOSIS NO.: 199396011606
Effects of site-specific acetylation on omega - conotoxin GVIA binding and function. 1993

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Structural studies of the calcium channel blocker omega - conotoxin and a partially active disulfide isomer. 1993

37/6/40 (Item 7 from file: 5) 08362270 BIOSIS NO.: 000094102793
SYNTHESIS AND CHARACTERIZATION OF A DISULFIDE BOND ISOMER OF OMEGA CONOTOXIN GVIA 1992

37/6/41 (Item 8 from file: 5) 08146972 BIOSIS NO.: 000042116395
STRUCTURE-ACTIVITY ANALYSIS OF OMEGA CONOTOXIN GVIA INTERACTION WITH NEURONAL CALCIUM CHANNELS 1991

37/6/42 (Item 9 from file: 5) 08113013 BIOSIS NO.: 000093112361
THE INHIBITION OF IODINE-125 OMEGA CONOTOXIN GVIA BINDING TO NEURONAL MEMBRANES BY NEOMYCIN MAY BE MEDIATED BY A GTP BINDING PROTEIN 1991

37/6/43 (Item 10 from file: 5) 07878216 BIOSIS NO.: 000041115714
HEMODYNAMIC EFFECTS OF OMEGA CONOTOXIN GVIA A N-TYPE CALCIUM CHANNEL BLOCKER IN NORMOTENSIVE AND SPONTANEOUSLY HYPERTENSIVE RATS 1991

37/6/44 (Item 11 from file: 5) 07785423 BIOSIS NO.: 000041071374
NMR SOLUTION STRUCTURE OF OMEGA CONOTOXIN GVIA 1991

37/6/45 (Item 12 from file: 5) 07676946 BIOSIS NO.: 000041021942
NEURAL RESPONSES IN GUINEA-PIG TRACHEA MEDIATED VIA OMEGA CONOTOXIN -SENSITIVE CALCIUM CHANNELS 1991

37/6/46 (Item 13 from file: 5) 07618789 BIOSIS NO.: 000091136673
DIFFERENTIAL SENSITIVITIES OF AVIAN AND MAMMALIAN NEUROMUSCULAR JUNCTIONS TO INHIBITION OF CHOLINERGIC TRANSMISSION BY OMEGA CONOTOXIN GVIA 1991

37/6/47 (Item 14 from file: 5) 07394813 BIOSIS NO.: 000040020472
IMMUNOLOCALIZATION OF OMEGA CONOTOXIN BINDING SITES AT THE FROG NEUROMUSCULAR JUNCTION 1990

37/6/48 (Item 15 from file: 5) 06974595 BIOSIS NO.: 000089086356
PRESYNAPTIC ALPHA-2-ADRENOCEPTOR AND KAPPA OPIATE RECEPTOR OCCUPANCY PROMOTES CLOSURE OF NEURONAL N-TYPE CALCIUM CHANNELS 1989

37/6/49 (Item 16 from file: 5) 06956124 BIOSIS NO.: 000089078131
OMEGA CONOTOXIN GVIA BLOCKS SYNAPTIC TRANSMISSION IN THE CA1 FIELD OF THE HIPPOCAMPUS 1989

37/6/50 (Item 17 from file: 5) 06686820 BIOSIS NO.: 000087129006
INTRASYNAPTOSOMAL FREE CALCIUM CONCENTRATION IS INCREASED BY PHORBOL ESTERS VIA A 1 4 DIHYDROPYRIDINE-SENSITIVE L-TYPE CALCIUM CHANNEL 1989

37/6/51 (Item 18 from file: 5) 06635464 BIOSIS NO.: 000087077627
EFFECTS OF SYNTHETIC OMEGA CONOTOXIN ON THE CONTRACTILE RESPONSES OF SEGMENTS OF RAT ILEUM STOMACH FUNDUS AND UTERUS AND GUINEA-PIG TAENIA COLI 1988

37/6/52 (Item 19 from file: 5) 06026458 BIOSIS NO.: 000035117821
A CALCIUM CHANNEL PROBE FOR HUMAN BRAIN SPECIFIC BINDING SITES FOR OMEGA CONOTOXIN 1988

37/6/53 (Item 20 from file: 5) 05883176 BIOSIS NO.: 000034106325
INTERACTION OF OMEGA CONOTOXIN WITH NEURONAL CALCIUM CHANNELS 1988

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BIOCHEMICAL STUDIES OF OMEGA CONOTOXIN GVIA A PEPTIDE TOXIN INHIBITING VOLTAGE-SENSITIVE CALCIUM CHANNELS 1987

37/6/55 (Item 22 from file: 5) 05847255 BIOSIS NO.: 000034070404
EFFECTS OF OMEGA CONOTOXIN IN ISOLATED RAT SUPERIOR CERVICAL GANGLIA 1987

37/6/56 (Item 23 from file: 5) 05626467 BIOSIS NO.: 000083099608
NEURONAL CALCIUM CHANNEL INHIBITORS SYNTHESIS OF OMEGA CONOTOXIN GVIA AND EFFECTS ON CALCIUM-45 UPTAKE BY SYNAPTOSOMES 1987

37/6/57 (Item 24 from file: 5)
05475771 BIOSIS NO.: 000033076624
EFFECT OF OMEGA CONOTOXIN ON THE CONTRACTILE RESPONSE OF RAT UTERINE MUSCLE 1987

37/6/58 (Item 25 from file: 5) 05304557 BIOSIS NO.: 000032027686
SYNTHESIS AND SECONDARY-STRUCTURE DETERMINATION OF OMEGA CONOTOXIN G VIA A 27 PEPTIDE WITH THREE INTRAMOLECULAR DISULFIDE BONDS 1986

37/7/6 (Item 6 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10765418 20363693 PMID: 10903496
Conotoxin TVIIA, a novel peptide from the venom of Conus tulipa 1. Isolation, characterization and chemical synthesis.
Hill JM; Atkins AR; Loughnan ML; Jones A; Adams DA; Martin RC; Lewis RJ; Craik DJ; Alewood PF
Centre for Drug Design and Development, The Institute for Molecular Bioscience, The University of Queensland, Brisbane, Queensland, Australia.
European journal of biochemistry (GERMANY) Aug 2000, 267 (15) p4642-8, ISSN 0014-2956 Journal Code: EMZ
Languages: ENGLISH Document type: Journal Article Record type: Completed
A novel conotoxin belonging to the 'four-loop' structural class has been isolated from the venom of the piscivorous cone snail Conus tulipa. It was identified using a chemical-directed strategy based largely on mass spectrometric techniques. The new toxin, conotoxin TVIIA, consists of 30 amino-acid residues and contains three disulfide bonds. The amino-acid sequence was determined by Edman analysis as SCSGRDSRCOOVCCMGLMCSRGKCVSIYGE where O = 4-transL-hydroxyproline. Two under-hydroxylated analogues, [Pro10]TVIIA and [Pro10,11]TVIIA, were also identified in the venom of C. tulipa. The sequences of TVIIA and [Pro10]TVIIA were further verified by chemical synthesis and coelution studies with native material. Conotoxin TVIIA has a six cysteine/four-loop structural framework common to many peptides from Conus venoms including the omega -, delta- and kappa-conotoxins. However, TVIIA displays little sequence homology with these well-characterized pharmacological classes of peptides, but displays striking sequence homology with conotoxin GS, a peptide from Conus geographus that blocks skeletal muscle sodium channels. These new toxins and GS share several biochemical features and represent a distinct subgroup of the four-loop conotoxins. Record Date Created: 20000928

37/7/11 (Item 11 from file: 155) DIALOG(R)File 155:MEDLINE(R)
08857093 94279504 PMID: 8010158
Different omega -conotoxins mark the development of Swiss Webster mouse cortex suggesting N-type voltage sensitive calcium channel subtypes.
Abbott JR; Litzinger MJ
Department of Pediatrics, University of Utah, Salt Lake City 84132.
International journal of developmental neuroscience (ENGLAND) Feb 1994, 12 (1) p43-7, ISSN 0736-5748 Journal Code: 126
Contract/Grant No.: HD 008867, HD, NICHD Languages: ENGLISH Document type: Journal Article Record type: Completed
omega -GVIA conotoxin has been used to mark presynaptic N-type voltage sensitive calcium channels (VSCC). Litzinger et al. used omega - conotoxin binding to describe a critical period of neurodevelopment in Swiss Webster mice between postnatal days (PND) 11 and 14, which appears to be important to the initiation of proper final development of the central nervous system. In this study, we compare how three different omega -conotoxins (i.e. GVIA from Conus geographus , MVIIA from Conus magus, and RVIA from Conus radiatus) mark N-type VSCC during this critical period in Swiss Webster mouse cortex. 125I-GVIA was bound to Swiss Webster mouse cortex synaptosomal membrane fractions at postnatal days 8 and 14. 125I-GVIA binding displacement curves were obtained by incubating membranes with increasing concentrations of unlabeled GVIA, MVIIA, and RVIA. Displacement curves and IC50 were calculated for each of these three omega -conotoxins, and then compared. At PND 14, GVIA, MVIIA and RVIA were able to displace greater than 95% of 125I-GVIA binding. At PND 8, however, MVIIA was only able to displace 83% of 125I-GVIA binding, and RVIA was only able to block 84%. The IC50 does not appear to change significantly during this period of development for any of the omega -conotoxins. The inability of MVIIA and RVIA to completely block 125I-GVIA binding in pre-critical period Swiss Webster cortex denotes an alteration in the composition of N-type VSCC binding sites. With this data, we have suggested the presence of subtypes of the N-type VSCC in the cortex of pre-critical period Swiss Webster mouse. Record Date Created: 19940721

38/6/1 (Item 1 from file: 5) 01351971 BIOSIS NO.: 000010092215
CONUS-AUSTRALIS AND CONUS- LATERCULATUS 1974

44/6/1 (Item 1 from file: 155) 11703969 21324260 PMID: 11430886
Autoradiographic localization of N-type VGCCs in gerbil hippocampus and failure of omega - conotoxin MVIIA to attenuate neuronal injury after transient cerebral ischemia. Jul 13 2001

44/6/2 (Item 2 from file: 155) 11679957 21243158 PMID: 11344322
Solution structure and backbone dynamics of an omega -conotoxin precursor. Mar 2001

44/6/3 (Item 3 from file: 155) 10759622 20213238 PMID: 10747778
Structural and dynamic characterization of omega - conotoxin MVIIA: the binding loop exhibits slow conformational exchange. Apr 11 2000

44/6/4 (Item 4 from file: 155) 10333464 99091279 PMID: 9876051

The effect of calcium channels blockers in the K+-evoked release of [3H]adenine nucleotides from rat brain cortical synaptosomes. Dec 11 1998

44/6/5 (Item 5 from file: 155) 10295622 98028723 PMID: 9359903
Analogies and differences between omega -conotoxins MVIIC and MVIID: binding sites and functions in bovine chromaffin cells. Dec 1997

44/6/6 (Item 6 from file: 155) 10154584 99278118 PMID: 10346894
Effects of chirality at Tyr13 on the structure-activity relationships of omega -conotoxins from Conus magus . May 25 1999

44/6/7 (Item 7 from file: 155) 09983819 99006668 PMID: 9792182
Pharmacotherapeutic potential of omega - conotoxin MVIIA (SNX-111), an N-type neuronal calcium channel blocker found in the venom of Conus magus Nov 1998

44/6/8 (Item 8 from file: 155) 09512398 95387290 PMID: 7658369
Characteristic features of inhibitory junction potentials evoked by single stimuli in the guinea-pig isolated taenia caeci. May 15 1995

44/6/9 (Item 9 from file: 155) 09510881 95055209 PMID: 7965828
The upregulation of acetylcholine release at endplates of alpha-bungarotoxin-treated rats: its dependency on calcium. Jul 1 1994

44/6/10 (Item 10 from file: 155) 09500121 95385787 PMID: 7656969
Solution structure of omega - conotoxin MVIIA using 2D NMR spectroscopy. Aug 21 1995

44/6/11 (Item 11 from file: 155) 09496095 95248539 PMID: 7731037
Solution structure of omega - conotoxin MVIIC, a high affinity ligand of P-type calcium channels, using 1H NMR spectroscopy and complete relaxation matrix analysis. Apr 21 1995

44/6/12 (Item 12 from file: 155) 09321851 97247935 PMID: 9094058
Incidence of serum anti-P/O-type and anti-N-type calcium channel autoantibodies in the Lambert-Eaton myasthenic syndrome. Mar 20 1997

44/6/13 (Item 13 from file: 155) 09106755 97042743 PMID: 8887942
Nitric oxide responsible for NMDA receptor-evoked inhibition of arachidonic acid incorporation into lipids of brain membrane. Sep 1996

44/6/14 (Item 14 from file: 155) 08894404 95239338 PMID: 7722641
Pharmacological dissection of multiple types of Ca2+ channel currents in rat cerebellar granule neurons. Apr 1995

44/6/15 (Item 15 from file: 155) 08887034 95094908 PMID: 8001657
Effect of omega -agatoxin-IVA on autonomic neurotransmission. Aug 11 1994

44/6/16 (Item 16 from file: 155) 08857093 94279504 PMID: 8010158
Different omega -conotoxins mark the development of Swiss Webster mouse cortex suggesting N-type voltage sensitive calcium channel subtypes. Feb 1994

44/6/17 (Item 17 from file: 155) 08735556 95239328 PMID: 7722635
Overexpression of potassium channel RNA: in vivo development rescues neurons from suppression of morphological differentiation in vitro. Apr 1995

44/6/18 (Item 18 from file: 155) 08628901 96018263 PMID: 7566366
Influence of Ca2+ channel modulators on [3H]purine release from rat cultured glial cells. Jun 1995

44/6/19 (Item 19 from file: 155) 08599232 95388123 PMID: 7659144
Three types of voltage-dependent calcium currents in cultured human neuroblastoma cells. Mar 1995

44/6/20 (Item 20 from file: 155) 08497086 95239312 PMID: 7722623
Cholinergic regulation of [Ca2+]i during cell division and differentiation in the mammalian retina. Apr 1995

44/6/160 (Item 160 from file: 155) 05548721 88224879 PMID: 2453348
Potassium depolarization elevates cytosolic free calcium concentration in rat anterior pituitary cells through 1,4-dihydropyridine-sensitive, omega - conotoxin -insensitive calcium channels. Jun 1988

44/6/165 (Item 165 from file: 155) 05478256 89266944 PMID: 2543080
Localization and mobility of omega - conotoxin -sensitive Ca2+ channels in hippocampal CA1 neurons. Jun 9 1989

44/6/166 (Item 166 from file: 155) 05473202 89165842 PMID: 2538116
Calcium channel binding in nerves and muscle of canine small intestine. Feb 28 1989

44/6/167 (Item 167 from file: 155) 05431951 90123648 PMID: 2692757
Contractions of rat aorta to endothelin are sensitive to nickel and cadmium ions but not nicardipine or omega - conotoxin . Dec 1989

44/6/168 (Item 168 from file: 155) 05402414 90222033 PMID: 2633190
Interaction of opiates with omega - conotoxin in guinea pig ileum in vitro. Nov-Dec 1989

44/6/169 (Item 1 from file: 5) 12860111 BIOSIS NO.: 200100067260
omega - conotoxin MVIIA: From marine snail venom to ana gesic drug. BOOK TITLE: Drugs from the sea 2000

44/6/170 (Item 2 from file: 5) 12640268 BIOSIS NO.: 200000393770
Synthesis and biological activity of 4-aminopiperidine derivatives as N-type calcium channel antagonists. 2000

44/6/171 (Item 3 from file: 5) 11718939 BIOSIS NO.: 199800500670
Synthesis of a non-peptide analogue of omega - conotoxin MVIIA. 1998

44/6/172 (Item 4 from file: 5) 10340307 BIOSIS NO.: 199698795225
Conotoxin -sensitive and conotoxin -resistant Ca-2+ currents in fish retinal ganglion cells. 1996

44/7/171 (Item 3 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2002 BIOSIS. All rts. reserv.
11718939 BIOSIS NO.: 199800500670
Synthesis of a non-peptide analogue of omega - conotoxin MVIIA.
AUTHOR: Menzler Stefan; Bikker Jack A; Horwell David C(a)
AUTHOR ADDRESS: (a)Parke-Davis Neurosci. Res. Cent., Robinson Way, Cambridge CB2 2QB**UK
JOURNAL: Tetrahedron Letters 39 (41):p7619-7622 Oct. 8, 1998 ISSN: 0040-4039 DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: An efficient synthesis of an alkylphenyl ether based peptidomimetic is described. The compound mimics three key amino acids of omega - conotoxin MVIIA from the cone shell Conus magus and may provide an entry to the design of low molecular weight antagonists of N-type neuronal calcium channels.

48/7/1 (Item 1 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10300097 98144527 PMID: 9483533
Activation of alpha 2-adrenoceptors causes inhibition of calcium channels but does not modulate inwardly-rectifying K+ channels in caudal raphe neurons.
Li YW; Bayliss DA
Department of Pharmacology, University of Virginia, Charlottesville 22908, USA.
Neuroscience (UNITED STATES) Feb 1998, 82 ' (3) p753-65, ISSN 0306-4522 Journal Code: NZR
Contract/Grant No.: NS33583, NS, NINDS Languages: ENGLISH Document type: Journal Article Record type: Completed
Many neurotransmitter receptors that interact with pertussis toxin-sensitive G proteins, including the alpha 2-adrenergic receptor, can modulate both voltage-dependent calcium channels and G protein-coupled inwardly-rectifying K+ channels. Serotonergic neurons of the medulla oblongata (nucleus raphe obscurus and nucleus raphe pallidus), which provide a major projection to sympathetic and motor output systems, receive a catecholaminergic input and express alpha 2-adrenergic receptors. Therefore, we tested the effects of norepinephrine on voltage-dependent calcium channels and G protein-coupled inwardly-rectifying K+ channels in neonatal raphe neurons using whole-cell recording in a brainstem slice preparation. Calcium channel currents were inhibited by norepinephrine in the majority of raphe neurons tested (88%) and in all identified tryptophan hydroxylase-immunoreactive (i.e. serotonergic) neurons. When tested in the same neurons, the magnitude of calcium current inhibition by norepinephrine (approximately 25%) was less than that induced by 5-hydroxytryptamine (approximately 50%). The norepinephrine-induced calcium current inhibition was mediated by alpha 2-adrenergic receptors; it was mimicked by UK 14304, an alpha 2-adrenergic receptor agonist and blocked by idazoxan, an alpha 2-adrenergic receptor antagonist, but not affected by prazosin or propanolol (alpha 1 and beta adrenergic antagonists, respectively). Calcium current inhibition by norepinephrine was essentially eliminated following application of omega- Conotoxin GVIA and omega-Agatoxin IVA, indicating that norepinephrine modulated N- and P/Q-type calcium channels predominantly. Calcium current inhibition by norepinephrine was voltage-dependent and mediated by pertussis toxin-sensitive G proteins. Thus, as expected, alpha 2-adrenergic receptor activation inhibited N- and P/Q-type calcium currents in medullary raphe neurons via pertussis toxin-sensitive G proteins. In parallel experiments, however, we found that norepinephrine had no effect on G protein-coupled inwardly-rectifying K+ channels in any raphe neurons tested, despite the robust activation of those channels in the same neurons by 5-hydroxytryptamine. Together, these data indicate that alpha 2-adrenergic receptors can modulate N- and P/Q-type calcium channels in caudal medullary raphe neurons but do not couple to the G protein-coupled inwardly-rectifying K+ channels which are also present in those cells. This is in contrast to the effect of 5-hydroxytryptamine1A receptor activation in caudal raphe neurons, and indicates a degree of specificity in the signalling by different pertussis toxin-sensitive G protein-coupled receptors to voltage-dependent calcium channels and G protein-coupled inwardly-rectifying K+ channels even within the same cell system. Record Date Created: 19980409

48/6/2 (Item 1 from file: 5) 11254621 BIOSIS NO.: 199800035953
Activation of alpha2-adrenoceptors causes inhibition of calcium channels but does not modulate inwardly-rectifying K+ channels in caudal raphe neurons. 1998

51/6/1 (Item 1 from file: 155) 11375220 21140940 PMID: 11246854
Variability in automated assignment of NOESY spectra and three-dimensional structure determination: a test case on three small disulfide-bonded proteins. Jan 2001

51/6/2 (Item 2 from file: 155) 10744765 98138433 PMID: 9477946
Three-dimensional solution structure of conotoxin psi-PIIIE, an acetylcholine gated ion channel antagonist. Feb 3 1998

51/6/3 (Item 3 from file: 155) 10743717 98104087 PMID: 9438859
Solution structure and proposed binding mechanism of a novel potassium channel toxin kappa- conotoxin PVIIA. Dec 15 1997

51/6/4 (Item 4 from file: 155) 10716335 20387358 PMID: 10818087
Single amino acid substitutions in kappa- conotoxin PVIIA disrupt interaction with the shaker K+ channel. Aug 11 2000

51/6/5 (Item 5 from file: 155) 09724241 98217295 PMID: 9548922
Three-dimensional structure of kappa- conotoxin PVIIA, a novel potassium channel-blocking toxin from cone snails. Apr 21 1998

51/6/6 (Item 6 from file: 155) 09631713 98079023 PMID: 9417043
kappa- Conotoxin PVIIA is a peptide inhibiting the shaker K+ channel. Jan 2 1998

51/6/7 (Item 7 from file: 155) 09538151 97383165 PMID: 9236004
A noncompetitive peptide inhibitor of the nicotinic acetylcholine receptor from Conus purpurascens venom. Aug 5 1997

51/6/8 (Item 8 from file: 155) 08609615 95403432 PMID: 7673220
A new family of Conus peptides targeted to the nicotinic acetylcholine receptor. Sep 22 1995

51/6/9 (Item 9 from file: 155) 08490325 95226378 PMID: 7711013
Purification, characterization, synthesis, and cloning of the lockjaw peptide from Conus purpurascens venom. Apr 18 1995

51/6/10 (Item 1 from file: 5) 13373995 BIOSIS NO.: 200200002816
kappa- Conotoxin PVIIA binding to Shaker K-channels and fast C-type inactivation are mutually exclusive. 2001

51/6/11 (Item 2 from file: 5) 13282690 BIOSIS NO.: 200100489839
delta- Conotoxin -PVIA affects voltage-dependent characteristics of sodium channels in frog sympathetic neurons. 2001

51/6/12 (Item 3 from file: 5) 13047812 BIOSIS NO.: 200100254961
Fast C-type inactivation abolishes the affinity of kappa- conotoxin PVIIA to Shaker K+-channels. 2001

51/6/13 (Item 4 from file: 5) 12731408 BIOSIS NO.: 200000484910
NMR and molecular modeling studies on the psi-conotoxins, non-competitive antagonists of the nicotinic acetylcholine receptor. 2000

51/6/14 (Item 5 from file: 5) 11940180 BIOSIS NO.: 199900186289
Blocking mechanism of kappa-PVIIA conotoxin on Shaker channels. 1999

51/6/15 (Item 6 from file: 5) 10596104 BIOSIS NO.: 199699217249
Kappa- conotoxin PVIIA, a Conus peptide targeted to potassium channels. 1996

51/6/16 (Item 7 from file: 5) 10385800 BIOSIS NO.: 199699006945
Strategy for rapid immobilization of prey by a fish-hunting marine snail. 1996

51/6/17 (Item 8 from file: 5) 10075785 BIOSIS NO.: 199598530703
Delta-Conotoxins, a family of subtype-specific Conus peptides which inhibit inactivation of voltage-sensitive sodium channels. 1995

53/6/1 (Item 1 from file: 155) 10764054 20320571 PMID: 10861378
The cyclic contryphan motif CPxXPXC, a robust scaffold potentially useful as an omega- conotoxin mimic. Sep 2000

53/6/2 (Item 2 from file: 155) 08857093 94279504 PMID: 8010158
Different omega-conotoxins mark the development of Swiss Webster mouse cortex suggesting N-type voltage sensitive calcium channel subtypes. Feb 1994

53/6/3 (Item 1 from file: 5) 12618923 BIOSIS NO.: 200000372425
The cyclic contryphan motif CPxXPXC, a robust scaffold potentially useful as an omega- conotoxin mimic. 2000

53/6/4 (Item 2 from file: 5) 09219432 BIOSIS NO.: 199497227802
Different omega-conotoxins mark the development of Swiss Webster mouse cortex suggesting N-type voltage sensitive calcium channel subtypes. 1994

53/7/1 (Item 1 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10764054 20320571 PMID: 10861378
The cyclic contryphan motif CPxXPXC, a robust scaffold potentially useful as an omega- conotoxin mimic.
Pallaghy PK; Norton RS
Biomolecular Research Institute, 343 Royal Parade, Parkville 3052, Australia. Paul.Pallaghy@bioresi.com.au
Biopolymers (UNITED STATES) Sep 2000, 54 (3) p173-9, ISSN 0006-3525 Journal Code: A5Z Languages: ENGLISH
Document type: Journal Article Record type: Completed
Contryphan-R, from venom of the cone-shell Conus radiatus , represents a novel cyclic peptide scaffold onto which residues may be grafted to mimic unrelated protein surfaces. Three substitutions were made at the x and X positions of the disulfide-bridged motif CPxXPXC, where X and x represent any L- and D-handed residues, respectively, P represents proline or hydroxyproline, and C a half-cystine. These substitutions were designed to mimic part of the pharmacophore of the unrelated globular polypeptide omega- conotoxin GVIA, which blocks N-type calcium channels. The structure of this engineered contryphan, YNK-contryphan-R ([D-Tyr4, Asn5, Lys7]contryphan-R), is shown to be similar to that of native contryphan-R (Pallaghy et al., Biochemistry, 1999, Vol. 38, pp. 13553-13559), confirming that the scaffold is robust with respect to the multiple substitutions. In particular, the alpha-beta bond vectors characterising the orientation of the side chains relative to the backbone are similar in contryphan-R, YNK-contryphan-R, and omega- conotoxin GVIA, which is the required result for a scaffold-based approach to molecular design. The solution structure of YNK-contryphan-R has an N-terminal, nonhydrogen-bonded, chain reversal centered on Hyp3-D-Trp4, and a C-terminal type I beta-turn. A minor form due to cis-trans isomerism of the Hyp2-Cys3 peptide bond is present in YNK-contryphan-R in a larger proportion than in contryphan-R. It is evident, particularly from the (3)J(HalphaHN) coupling constants, that YNK-contryphan-R is more flexible than contryphan-R, probably due to the absence in YNK-contryphan-R of the Pro-Trp packing present in the native molecule. Nevertheless, the structure confirms that cyclic peptide molecular designs can achieve the intended conformations. Copyright 2000 John Wiley & Sons, Inc. Record Date Created: 20000828

53/7/2 (Item 2 from file: 155) DIALOG(R)File 155:MEDLINE(R)
08857093 94279504 PMID: 8010158
Different omega-conotoxins mark the development of Swiss Webster mouse cortex suggesting N-type voltage sensitive calcium channel subtypes.
Abbott JR; Litzinger MJ
Department of Pediatrics, University of Utah, Salt Lake City 84132.
International journal of developmental neuroscience (ENGLAND) Feb 1994, 12 (1) p43-7, ISSN 0736-5748 Journal Code: 126 Contract/Grant No.: HD 008867, HD, NICHD Languages: ENGLISH Document type: Journal Article Record type: Completed
omega-GVIA conotoxin has been used to mark presynaptic N-type voltage sensitive calcium channels (VSCC). Litzinger et al. used omega- conotoxin binding to describe a critical period of neurodevelopment in Swiss Webster mice between postnatal days (PND) 11 and 14, which appears to be important to the initiation of proper final development of the central nervous system. In this study, we compare how three different omega-conotoxins (i.e. GVIA from Conus geographus, MVIIA from Conus magus, and RVIA from Conus radiatus) mark N-type VSCC during this critical period in Swiss Webster mouse cortex. 125I-GVIA was bound to Swiss Webster mouse cortex synaptosomal membrane fractions at postnatal days 8 and 14. 125I-GVIA binding displacement curves were obtained by incubating membranes with increasing concentrations of unlabeled GVIA, MVIIA, and RVIA. Displacement curves and IC50 were calculated for each of these three omega-conotoxins, and then compared. At PND 14, GVIA, MVIIA and RVIA were able to displace greater than 95% of 125I-GVIA binding. At PND 8, however, MVIIA was only able to displace 83% of 125I-GVIA binding, and RVIA was only able to block 84%. The IC50 does not appear to change significantly during this period of development for any of the omega-conotoxins. The inability of MVIIA and RVIA to completely block 125I-GVIA binding in pre-critical period Swiss Webster cortex denotes an alteration in the composition of N-type VSCC binding sites. With this data, we have suggested the presence of subtypes of the N-type VSCC in the cortex of pre-critical period Swiss Webster mouse. Record Date Created: 19940721

60/6/1 (Item 1 from file: 155) 11783856 21540680 PMID: 11683628
delta- Conotoxin Structure/Function through a Cladistic Analysis. Nov 6 2001

60/6/2 (Item 2 from file: 155) 09935906 99036623 PMID: 9819194
An O-glycosylated neuroexcitatory conus peptide. Nov 17 1998

60/6/3 (Item 3 from file: 155) 08463985 95138099 PMID: 7836370
A new conotoxin affecting sodium current inactivation interacts with the delta- conotoxin receptor site. Jan 20 1995

60/6/4 (Item 4 from file: 155) 08314289 95103030 PMID: 7804605
Calcium channel subtypes in rat brain: biochemical characterization of the high-affinity receptors for omega-conopeptides SNX-230 (synthetic MVIIC), SNX-183 (SVIB), and SNX-111 (MVIIA). Aug 1994

60/6/5 (Item 5 from file: 155) 08110203 94132020 PMID: 8300586
A new neurotoxin receptor site on sodium channels is identified by a conotoxin that affects sodium channel inactivation in molluscs and acts as an antagonist in rat brain. Jan 28 1994

60/6/6 (Item 6 from file: 155) 06887472 93003172 PMID: 1390774
Novel alpha- and omega-conotoxins from Conus striatus venom. Oct 20 1992

60/6/7 (Item 7 from file: 155) 05219870 89062448 PMID: 3196703
Phylogenetic specificity of cholinergic ligands: alpha- conotoxin SI. Sep 6 1988

60/6/8 (Item 1 from file: 5) 12614201 BIOSIS NO.: 200000367703
Solution structure of alpha- conotoxin SI. 2000

60/6/9 (Item 2 from file: 5) 10583622 BIOSIS NO.: 199699204767
Effects of ibogaine and noribogaine on phosphoinositide hydrolysis. 1996

60/6/10 (Item 3 from file: 5) 10348684 BIOSIS NO.: 199698803602
Neuroactive peptides of the marine snail, Conus striatus . 1996

60/6/11 (Item 4 from file: 5) 10345420 BIOSIS NO.: 199698800338
Scorpion toxins affecting sodium current inactivation bind to distinct homologous receptor sites on rat brain and insect sodium channels. 1996

60/6/12 (Item 5 from file: 5) 10071539 BIOSIS NO.: 199598526457
A new family of Conus peptides targeted to the nicotinic acetylcholine receptor. 1995

60/6/13 (Item 6 from file: 5) 07872222 BIOSIS NO.: 000092131588
ALPHA CONOTOXINS SMALL PEPTIDE PROBES OF NICOTINIC ACETYLCHOLINE RECEPTORS 1991

60/7/3 (Item 3 from file: 155) DIALOG(R)File 155:MEDLINE(R) 08463985 95138099 PMID: 7836370
A new conotoxin affecting sodium current inactivation interacts with the delta- conotoxin receptor site.
Fainzilber M; Lodder JC; Kits KS; Kofman O; Vinnitsky I; Van Rietschoten J; Zlotkin E; Gordon D
Department of Cell and Animal Biology, Silberman Institute of Life Sciences, Hebrew University of Jerusalem, Israel.
Journal of biological chemistry (UNITED STATES) Jan 20 1995, 270 (3) p1123-9, ISSN 0021-9258 Journal Code: HIV
Languages: ENGLISH Document type: Journal Article Record type: Completed
We describe a new peptide conotoxin affecting sodium current inactivation, that competes on binding with delta- conotoxin TxVIA (delta TxVIA). The amino acid sequence of the new toxin, designated conotoxin NgVIA (NgVIA), is SKCFSOGTFCGIKOGLCCSVRCFSLFCISFE (where O is trans-4-hydroxyproline). The primary structure of NgVIA has an identical cysteine framework and similar hydrophobicity as delta TxVIA but differs in its net charge. NgVIA competes with delta TxVIA on binding to rat brain synaptosomes and molluscan central nervous system and strongly inhibits sodium current inactivation in snail neurons, as does delta TxVIA. In contrast to delta TxVIA, NgVIA is a potent paralytic toxin in vertebrate systems, its binding appears to be voltage-dependent, and it synergically increases veratridine-induced sodium influx to rat brain synaptosomes. delta TxVIA acts as a partial antagonist to NgVIA in rat brain in vivo. NgVIA appears to act via a receptor site distinct from that of delta TxVIA but similar to that of Conus striatus toxin. This new toxin provides a lead for structure-function relationship studies in the delta-conotoxins and will enable analysis of the functional significance of this complex of receptor sites in gating mechanisms of sodium channels. Record Date Created: 19950224

60/7/4 (Item 4 from file: 155) DIALOG(R)File 155:MEDLINE(R)
08314289 95103030 PMID: 7804605
Calcium channel subtypes in rat brain: biochemical characterization of the high-affinity receptors for omega-conopeptides SNX-230 (synthetic MVIIC), SNX-183 (SVIB), and SNX-111 (MVIIA).
Woppmann A; Ramachandran J; Miljanich GP
Neurex Corporation, Menlo Park, California 94025-1012.
Molecular and cellular neurosciences (UNITED STATES) Aug 1994, 5 (4) p350-7, ISSN 1044-7431 Journal Code: B1D
Languages: ENGLISH Document type: Journal Article Record type: Completed
High-threshold voltage-sensitive calcium channels of the N-type, L-type, and P-type have been distinguished in the mammalian CNS predominantly on the basis of their sensitivity to selective antagonists. Matching them with genes identified by molecular cloning is an ongoing undertaking. Whereas L-type channels are characterized by their sensitivity to dihydropyridines and P-type channels by sensitivity to the funnel-web spider toxin AgaIVA, the N-type channel has been shown to be recognized by the omega-conopeptides GVIA and MVIIA. Recently, two new members of the family of omega-conopeptides--MVIIC from the marine snail Conus magus and SVIB from Conus striatus --have been described. Binding and electrophysiological data suggest that these two peptides, in addition to interacting with N-type calcium channels, interact with a widely distributed receptor in neuronal membranes that is distinct from N-type channels. In this report we demonstrate through biochemical and pharmacological differentiation at individual receptor polypeptide resolution, by affinity cross-linking, SDS-PAGE, and autoradiography, that SNX-230 (synthetic MVIIC) binds with high affinity to a calcium channel alpha 1 subunit distinct from the high-affinity alpha 1 target of SNX-111 (synthetic MVIIA). SNX-183 (synthetic SVIB) interacts with both alpha 1 subunits with lower affinity. Whereas the alpha 1 subunit recognized with high affinity by MVIIA corresponds to the N-type channel, the other represents a novel calcium channel distinct from N-, L-, and perhaps P-type channels. Record Date Created: 19950130

60/7/6 (Item 6 from file: 155) DIALOG(R)File 155:MEDLINE(R)
06887472 93003172 PMID: 1390774
Novel alpha- and omega-conotoxins from Conus striatus venom.
Ramilo CA; Zafaralla GC; Nadasdi L; Hammerland LG; Yoshikami D; Gray WR; Kristipati R; Ramachandran J; Miljanich G; Olivera BM; et al
Marine Science Institute, University of Philippines, Diliman, Quezon City.
Biochemistry (UNITED STATES) Oct 20 1992, 31 (41) p9919-26, ISSN 0006-2960 Journal Code: A0G Contract/Grant No.: GM 22737, GM, NIGMS; GM 34913, GM, NIGMS Languages: ENGLISH Document type: Journal Article Record type: Completed
Three neurotoxic peptides from the venom of Conus striatus have been purified, biochemically characterized, and chemically synthesized. One of these, an acetylcholine receptor blocker designated alpha- conotoxin SII, has the sequence GCCCNPACGPNYGCGTSCS. In contrast to all other alpha-conotoxins, SII has three disulfide bonds (instead of two), has no net positive charge, and has a free C-terminus. The other two paralytic peptides are Ca channel-targeted omega-conotoxins, SVIA and SVIB. omega-SVIA is the smallest natural omega- conotoxin so far characterized and has the sequence CRSSGSPCGVTSICCGRCYRGKCT-NH2. Although omega- conotoxin SVIA is a potent paralytic toxic in lower vertebrate species, it was much less effective in mammals. The third toxin, omega- conotoxin SVIB, has the sequence CKLKGQSCRKTSYDCCSGSCGRSGKC-NH2. This peptide has a different pharmacological specificity from other omega-conotoxins previously purified from Conus venoms; only omega- conotoxin SVIB has proven to be lethal to mice upon ic injection. Binding competition experiments with rat brain synaptosomal membranes indicate that the high-affinity binding site for omega- conotoxin SVIB is distinct from the high-affinity omega- conotoxin GVIA or MVIIA site. Record Date Created: 19921118

60/7/10 (Item 3 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2002 BIOSIS. All rts. reserv.
10348684 BIOSIS NO.: 199698803602
Neuroactive peptides of the marine snail, Conus striatus .
AUTHOR: Cruz L J
AUTHOR ADDRESS: Marine Sci. Inst., Univ. Philippines, Quezon City** Philippines
JOURNAL: Journal of Natural Toxins 5 (1):p122 1996 CONFERENCE/MEETING: 209th American Chemical Society National Meeting on Natural Toxins Anaheim, California, USA April 2, 1995-April 7, 1996 ISSN: 1058-8108 RECORD TYPE: Citation
LANGUAGE: English

65/6/1 (Item 1 from file: 155) 10804036 99254114 PMID: 10318957
A conotoxin from Conus textile with unusual posttranslational modifications reduces presynaptic Ca2+ influx. May 11 1999

65/6/2 (Item 2 from file: 155) 10523835 20146306 PMID: 10679974
Structure determination of two conotoxins from Conus textile by a combination of matrix-assisted laser desorption/ionization time-of-fl and electrospray ionization mass spectrometry and biochemical methods. Feb 2000

65/6/3 (Item 3 from file: 155) 10506439 20143473 PMID: 10677206
The spasmodic peptide defines a new conotoxin superfamily. Feb 22 2000

65/6/4 (Item 4 from file: 155) 10386606 20014562 PMID: 10545191
Hydrophobic amino acids define the carboxylation recognition site in the precursor of the gamma-carboxyglutamic-acid-containing conotoxin epsilon-TxIX from the marine cone snail Conus textile . Nov 2 1999

65/6/5 (Item 5 from file: 155) 09663517 98145210 PMID: 9484216
gamma- Conotoxin -PnVIIA, a gamma-carboxyglutamate-containing peptide agonist of neuronal pacemaker cation currents. Feb 10 1998

65/6/6 (Item 6 from file: 155) 09523191 97022130 PMID: 8868490
Mass spectrometric-based revision of the structure of a cysteine-rich peptide toxin with gamma-carboxyglutamic acid, TxVIIA, from the sea snail, Conus textile . Mar 1996

65/6/7 (Item 7 from file: 155) 09045166 97085337 PMID: 8931478
Interactions of delta-conotoxins with alkaloid neurotoxins reveal differences between the silent and effective binding sites on voltage-sensitive sodium channels. Dec 1996

65/6/8 (Item 8 from file: 155) 08930856 96266175 PMID 8679638
A novel hydrophobic omega- conotoxin blocks molluscan dihydropyridine-sensitive calcium channels. Jul 2 1996

65/6/9 (Item 9 from file: 155) 08555082 95332960 PMID: 7608772
Alterations of voltage-activated sodium current by a novel conotoxin from the venom of Conus gloriamaris. Mar 1995

65/6/10 (Item 10 from file: 155) 06832547 90214607 PMID: 1691090
Constant and hypervariable regions in conotoxin propeptides. Apr 1990

65/6/11 (Item 1 from file: 5) 12391064 BIOSIS NO.: 200000144566
Two novel hyperactivity causing Conus textile peptides. 1999

65/6/12 (Item 2 from file: 5) 12228223 BIOSIS NO.: 199900523072
Synthesis, bioactivity, and cloning of the L-type calcium channel blocker omega- conotoxin TxVII. 1999

65/6/13 (Item 3 from file: 5) 10345420 BIOSIS NO.: 199698800338
Scorpion toxins affecting sodium current inactivation bind to distinct homologous receptor sites on rat brain and insect sodium channels. 1996

65/6/14 (Item 4 from file: 5) 09219327 BIOSIS NO.: 199497227697
A toxin from the venom of the predator snail Conus textile modulates ionic currents in Aplysia bursting pacemaker neuron. 1994

65/6/15 (Item 5 from file: 5) 09173865 BIOSIS NO.: 199497182235
A New Neurotoxin Receptor Site on Sodium Channels Is Identified by a Conotoxin That Affects Sodium Channel Inactivation in Molluscs and Acts as an Antagonist in Rat Brain. 1994

65/6/16 (Item 6 from file: 5) 07974006 BIOSIS NO.: 000093041584
MOLLUSC-SPECIFIC TOXINS FROM THE VENOM OF CONUS- TEXTILE –NEOVICARIUS 1991

65/7/1 (Item 1 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10804036 99254114 PMID: 10318957
A conotoxin from Conus textile with unusual posttranslational modifications reduces presynaptic Ca2+ influx.
Rigby AC; Lucas-Meunier E; Kalume DE; Czerwiec E; Hambe B; Dahlqvist I; Fossier P; Baux G; Roepstorff P; Baleja JD; Furie BC; Furie B; Stenflo J
Marine Biological Laboratory, Woods Hole, MA 02543, USA.
Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) May 11 1999, 96 (10) p5758-63, ISSN 0027-8424 Journal Code: PV3 Languages: ENGLISH Document type: Journal Article Record type: Completed
Cone snails are gastropod mollusks of the genus Conus that live in tropical marine habitats. They are predators that paralyze their prey by injection of venom containing a plethora of small, conformationally constrained peptides (conotoxins). We report the identification, characterization, and structure of a gamma-carboxyglutamic acid-containing peptide, conotoxin epsilon-TxIX, isolated from the venom of the molluscivorous cone snail, Conus textile . The disulfide bonding pattern of the four cysteine residues, an unparalleled degree of posttranslational processing including bromination, hydroxylation, and glycosylation define a family of conotoxins that may target presynaptic Ca2+ channels or act on G protein-coupled presynaptic receptors via another mechanism. This conotoxin selectively reduces neurotransmitter release at an Aplysia cholinergic synapse by reducing the presynaptic influx of Ca2+ in a slow and reversible fashion. The three-dimensional structure, determined by two-dimensional 1H NMR spectroscopy, identifies an electronegative patch created by the side chains of two gamma-carboxyglutamic acid residues that extend outward from a cavernous cleft. The glycosylated threonine and hydroxylated proline enclose a localized hydrophobic region centered on the brominated tryptophan residue within the constrained intercysteine region. Record Date Created: 19990617

65/7/2 (Item 2 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10523835 20146306 PMID: 10679974
Structure determination of two conotoxins from Conus textile by a combination of matrix-assisted laser desorption/ionization time-of-flight and electrospray ionization mass spectrometry and biochemical methods.
Kalume DE; Stenflo J; Czerwiec E; Hambe B; Furie BC; Furie B; Roepstorff P
Department of Molecular Biology, University of Southern Denmark, Odense University, Campusvej 55, DK-5230 Odense M, Denmark.
Journal of mass spectrometry (ENGLAND) Feb 2000, 35 (2) p145-56, ISSN 1076-5174 Journal Code: CMB Languages: ENGLISH Document type: Journal Article Record type: Completed
Two highly modified conotoxins from the mollusc Conus textile , epsilon-TxIX and Gla(1)-TxVI, were characterized by matrix-assisted laser desorption/ionization and electrospray mass spectrometry and also by electrospray ionization tandem and triple mass spectrometry in combination with enzymatic cleavage and chemical modification reactions. The mass spectrometric studies allowed the confirmation of the sequence determined by Edman degradation and assignment of unidentified amino acid residues, among which bromotryptophan residues and an O-glycosylated threonine residue were observed. Methyl esterification was found necessary for the site-specific assignment of the Gla residues in the peptides. Copyright 2000 John Wiley & Sons, Ltd. Record Date Created: 20000328

65/7/3 (Item 3 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10506439 20143473 PMID: 10677206
The spasmodic peptide defines a new conotoxin superfamily.
Lirazan MB; Hooper D; Corpuz GP; Ramilo CA; Bandyopadhyay P; Cruz LJ; Olivera BM
Department of Biology, University of Utah, Salt Lake City, Utah 84112, USA.
Biochemistry (UNITED STATES) Feb 22 2000, 39 (7) p1583-8, ISSN 0006-2960 Journal Code: A0G Contract/Grant No.: GM48677, GM, NIGMS Languages: ENGLISH Document type: Journal Article Record type: Completed
We purified and characterized a peptide from the venom of Conus textile Record Date Created: 20000313

65/7/8 (Item 8 from file: 155) DIALOG(R)File 155:MEDLINE(R)
08930856 96266175 PMID: 8679638
A novel hydrophobic omega- conotoxin blocks molluscan dihydropyridine-sensitive calcium channels.
Fainzilber M; Lodder JC; van der Schors RC; Li KW; Yu Z; Burlingame AL; Geraerts WP; Kits KS
Graduate School Neurosciences Amsterdam, Institute of Neuroscience, Vrije Universiteit, The Netherlands.
Biochemistry (UNITED STATES) Jul 2 1996, 35 (26) p8748-52, ISSN 0006-2960 Journal Code: A0G Contract/Grant No.: RR01614, RR, NCRR Languages: ENGLISH Document type: Journal Article Record type: Completed
A novel calcium channel blocking peptide designated omega- conotoxin -Tx VII has been characterized from the venom of the molluscivorous snail Conus textile . The amino acid sequence (CKQADEPCDVFSLDCCTGICLGVCMW) reveals the characteristic cysteine framework of omega-conotoxins, but it is extremely hydrophobic for this pharmacological class of peptides and further unusual in its net negative charge (-3). It is further striking that the sequence of TxVII, a calcium current blocker, is 58% identical to that of delta- conotoxin -TxVIA, which targets sodium channels. TxVII effects were examined in the caudodorsal cell (CDC) neurons from the mollusc Lymnaea stagnalis. The toxin has no significant effect on sodium or potassium currents in these cells, but it clearly blocks the calcium currents. TxVII most prominently blocks the slowly inactivating, dihydropyridine-(DHP-) sensitive current in CDCs, while blockade of the rapidly inactivating current is less efficient. This novel omega- conotoxin is apparently targeted to DHP-sensitive calcium channels and thereby provides a lead for future design of selective conopeptide probes for L-type channels. Record Date Created: 19960822

65/7/11 (Item 1 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2002 BIOSIS. All rts. reserv.
12391064 BIOSIS NO.: 200000144566
Two novel hyperactivity causing Conus textile peptides.
AUTHOR: Lirazan M B(a); Craig A G; Olivera B M; Hooper D; McIntosh J M; Cruz L J
AUTHOR ADDRESS: (a)Dept. of Phys. Sciences and Math., U. P. Manila, Manila **Philippines
JOURNAL: Society for Neuroscience Abstracts. 25 (1-2):p962 1999 CONFERENCE/MEETING: 29th Annual Meeting of the Society for Neuroscience. Miami Beach, Florida, USA. October 23-28, 1999 SPONSOR: Society for Neuroscience
ISSN: 0190-5295 RECORD TYPE: Citation LANGUAGE: English SUMMARY LANGUAGE: English

65/7/12 (Item 2 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2002 BIOSIS. All rts. reserv.
12228223 BIOSIS NO.: 199900523072
Synthesis, bioactivity, and cloning of the L-type calcium channel blocker omega- conotoxin TxVII.
AUTHOR: Sasaki Toru; Feng Zhong-Ping; Scott Randolph; Grigoriev Nikita; Syed Naweed I; Fainzilber Michael; Sato Kazuki(a)
AUTHOR ADDRESS: (a)Mitsubishi Kasei Institute of Life Sciences, 11 Minamiooya, Machida-shi, Tokyo, 194-8511**Japan
JOURNAL: Biochemistry 38 (39):p12876-12884 Sept. 28, 1999 ISSN: 0006-2960 DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English SUMMARY LANGUAGE: English
ABSTRACT: omega- Conotoxin TxVII is the first conotoxin reported to block L-type currents. In contrast to other omega-conotoxins, its sequence is characterized by net negative charge and high hydrophobicity, although it retains the omega-conotoxin cysteine framework. In order to obtain structural information and to supply material for further characterization of its biological function, we synthesized TxVII and determined its disulfide bond pairings. Because a linear precursor with free SH groups showed a strong tendency to aggregate and to polymerize, we examined many different conditions for air oxidation and concluded that a mixture of cationic buffer and hydrophobic solvent was the most effective for the folding of TxVII. Synthetic TxVII was shown to suppress the slowly inactivating voltage-dependent calcium current in cultured Lymnaea RPeD1 neurons and furthermore to suppress synaptic transmission between these neurons and their follower cells. In contrast, TxVII did not block calcium flux through L-type channels in PC12 cells, suggesting a phyletic or subtype specificity in this channel family. Disulfide bond pairings of TxVII and its isomers were determined by enzymatic fragmentation in combination with chemical synthesis, thus revealing that TxVII has the same disulfide bond pattern as other omega-conotoxins. Furthermore, the CD spectrum of TxVII is similar to those of omega-conotoxins MVIIA and MVIIC. The precursor sequence of TxVII was determined by cDNA cloning and shown to be closest to that of delta- conotoxin TxVIA, a sodium channel inactivation inhibitor. Thus TxVII conserves the structural fold of other omega-conotoxins, and the TxVIA/TxVII branch of this family reveals the versatility of its structural scaffold, allowing evolution of structurally related peptides to target different channels.

68/6/1 (Item 1 from file: 155) 10765419 20363694 PMID: 10903497
Conotoxin TVIIA, a novel peptide from the venom of Conus tulipa 2. Three-dimensional solution structure. Aug 2000

68/6/2 (Item 2 from file: 155) 10765418 20363693 PMID: 10903496
Conotoxin TVIIA, a novel peptide from the venom of Conus tulipa 1. Isolation, characterization and chemical synthesis. Aug 2000

68/6/3 (Item 3 from file: 155) 09247488 97135949 PMID: 8981486
Inhibition of calcium channels in rat hippocampal CA1 neurons by conantokin-T. Dec 13 1996

68/6/4 (Item 1 from file: 5) 11151607 BIOSIS NO.: 199799772752
Identification of two novel conotoxin targets: Uptake-1 and the alpha-1-adrenoceptor. 1997

68/6/5 (Item 2 from file: 5) 09987056 BIOSIS NO.: 199598441974
Behavioral effects of the selective N-type neuronal calcium channel antagonist SNX-185 (omega- conotoxin TVIA) in mice. 1995

68/7/2 (Item 2 from file: 155) DIALOG(R)File 155:MEDLINE(R)
10765418 20363693 PMID: 10903496
Conotoxin TVIIA, a novel peptide from the venom of Conus tulipa 1. Isolation, characterization and chemical synthesis.
Hill JM; Atkins AR; Loughnan ML; Jones A; Adams DA; Martin RC; Lewis RJ; Craik DJ; Alewood PF
Centre for Drug Design and Development, The Institute for Molecular Bioscience, The University of Queensland, Brisbane, Queensland, Australia.
European journal of biochemistry (GERMANY) Aug 2000, 267 (15) p4642-8, ISSN 0014-2956 Journal Code: EMZ Languages: ENGLISH Document type: Journal Article Record type: Completed
A novel conotoxin belonging to the 'four-loop' structural class has been isolated from the venom of the piscivorous cone snail Conus tulipa . It was identified using a chemical-directed strategy based largely on mass spectrometric techniques. The new

toxin, conotoxin TVIIA, consists of 30 amino-acid residues and contains three disulfide bonds. The amino-acid sequence was determined by Edman analysis as SCSGRDSRCOOVCCMGLMCSRGKCVSIYGE where O = 4-transL-hydroxyproline. Two under-hydroxylated analogues, [Pro10]TVIIA and [Pro10,11]TVIIA, were also identified in the venom of C. tulipa . The sequences of TVIIA and [Pro10]TVIIA were further verified by chemical synthesis and coelution studies with native material. Conotoxin TVIIA has a six cysteine/four-loop structural framework common to many peptides from Conus venoms including the omega-, delta- and kappa-conotoxins. However, TVIIA displays little sequence homology with these well-characterized pharmacological classes of peptides, but displays striking sequence homology with conotoxin GS, a peptide from Conus geographus that blocks skeletal muscle sodium channels. These new toxins and GS share several biochemical features and represent a distinct subgroup of the four-loop conotoxins. Record Date Created: 20000928